A RETROSPECTIVE STUDY OF ORBITAL FRACTURES

DISSERTATION SUBMITTED FOR

MASTER OF SURGERY DEGREE

BRANCH – III- OPHTHALMOLOGY

APRIL 2013

THE TAMILNADU

DR. M.G.R MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

Dept. Of Ophthalmology

Govt. Rajaji Hospital

Madurai

CERTIFICATE

This is to certify that this dissertation entitled **"A RETROSPECTIVE STUDY OF ORBITAL FRACTURES"** has been done under my guidance in the Department of OPHTHALMOLOGY, MADURAI MEDICAL COLLEGE, MADURAI.

I Certify regarding the authenticity of the work done to prepare this dissertation.

Dr.P.THIYAGARJAN , M.S.,D.O.,

PROFESSOR & H.O.D

Dept. Of Ophthalmology

GOVT. RAJAJI HOSPITAL

MADURAI MEDICAL COLLEGE,

MADURAI.

<u>DECLARATION</u>

I, **Dr.R. NITHYA** declare that, I carried out this work on **"A RETROSPECTIVE STUDY OF ORBITAL FRACTURES"**at Govternment Rajaji Hospital, Madurai, I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, or diploma to any other University, Board, either in India or abroad.

This is submitted to the Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S Degree examination in Ophthalmology.

Place: Madurai **Dr. R.NITHYA**

Date :

ACKNOWLEDGEMENT

I am grateful to The **Dean, Dr.N.Mohan M.S.**, Madurai Medical College and Government Rajaji Hospital, Madurai for permitting me to carry out this study.

I extend my sincere thanks to **Prof.Dr.P.Thiyagarajan,MS.,DO, Head of the Department of Ophthalmology,** for his valuable guidance and support during the study period.

I would like to express my sincere thanks and deep sense of gratitude to **Prof. Dr.G.S.Srinivasan,MS.,DO** for his constant help, guidance and encouragement given to me throughout this study.

I am highly indebted to **Prof. Dr.N.Parvathasundari,MS.,DO** whose sincere guidance and encouragement were a source of strengthfor the successful completion of this study.

I express my sincere thanks to **Prof. Dr.N.Muthukumar,Mch, Professor of Neurosurgery, Prof. Dr. C.Balasubramanian,MS.,Mch,** H.O.D, Department of Plastic Surgery and **Prof. Dr.R.Anitha,MDS, Professor**

of Faciomaxillary Surgery for their valuable suggestions and support given to me throughout the study.

CONTENTS

INTRODUCTION

Orbital fractures occurs in a significant number of patients presenting with blunt trauma to the face and the skull. It may be either limited to the boundaries of the orbit itself or associated with extensive fractures of the craniofacial skeleton. Orbital fractures are of ophthalmological importance as the orbit lodges the eye and soft tissue which supports the globe and may suffer from direct or indirect injury.

Complications like globe rupture, malposition of the globe, orbital haemorrhage, direct or indirect optic nerve injury or injury to the lacrimal gland are the serious complications which can occur after orbital fracture.

The long term sequelae of orbital fractures are functional impairment and aesthetic deformity. Therefore every ophthalmologist should be familiar with the evaluation and management of blunt orbital trauma and orbital fractures.[22,67,76]

The management of orbital fractures has improved a lot during the last decade due to increased use of rigid internal fixation of craniofacial fractures including many of those which involve the orbit.[33,53,54,77,80]

SURGICAL ANATOMY OF THE ORBIT

The bony orbit is formed by seven bones that also form part of the facial skeleton and cranial cavity.[88] The medial walls of the orbit are parallel to each other and are separated by ethmoid and sphenoid sinuses. The lateral walls are at an angle of 45° to the medial walls which separate the orbit from muscular temporal fossa anteriorly and from the middle cranial fossa posteriorly . The orbit lies above the maxillary sinuses and beneath the anterior cranial fossa. The orbit is shaped like a pyramid opening at its base i.e orbital rims and apex at the lesser wing of sphenoid bone. The optic nerve exits the orbit at the apex through the optic foramen. The anteroposterior depth of the orbit is 40 to 50 mm from the inferior orbital margin to the apex. The base contributed by orbital rim and apex formed by sphenoid bone is relatively strong but the middle portion is formed by thin bones. This is particularly true of floor and medial wall of the orbit.[21,22]

ROOF OF THE ORBIT: Formed by

- Orbital plate of the frontal bone
- Lesser wing of the sphenoid bone

The roof lies beneath the anterior cranial fossa and frontal sinus. The lacrimal gland is situated in the fossa formed by anterolateral margin of the frontal bone. Trochlear fossa is situated in the anteromedial aspect, about 5 mm from its margin over which tendinous pulley of the superior oblique muscle inserts. The optic foramen lies at the apex of the roof of the orbit. The supraorbital notch which transmits the supraorbital nerve which is a branch of the frontal division of the trigeminal nerve lies at the superior margin of the frontal bone.

The roof is strong usually and only rarely blunt trauma can explode it. However,small dehiscences are not uncommon in the orbital roof. Its incidence rate is about 15%.[86,89] The optic nerve pierces the roof from the midline to form optic foramina at an angle of about 35°. The meningolacrimal foramen is situated near the suture between the frontal and the sphenoid bones about 30 mm posterior to the orbital rim. It conducts an anastomosis between the middle meningeal artery and the root of lacrimal artery in approximately 30% of the individuals.[20]

MEDIAL WALL OF THE ORBIT: Formed by

- Body of the sphenoid bone
- Ethmoid bone
- Frontal process of the maxilla
- Lacrimal bone

Medial wall is the smallest and thinnest of the orbital walls. The lacrimal sac fossa composed of frontal process of the maxilla and the lacrimal bone is located in the anterior aspect of medial orbit and contains lacrimal sac drainage apparatus. The inflammation in ethmoid sinus spreads readily to the orbit through lamina papyracea of the ethmoid bone which is the thinnest bone of the orbit. The bulla of ethmoid pneumatisation is seen as a honey comb pattern beneath the lamina papyracea. This supportive structure explains why medial wall fractures less often than the thicker orbital floor.

There are some congenital dehiscences at the ethmoidal sutural lines and age atrophy is seen centrally in the ethmoid plate. The anterior and posterior ethmoidal foramina are located at the frontoethmoidal suture approximately 24 mm and 36 mm posterior to the anterior lacrimal crest. It transmits branches of nasociliary nerve and ophthalmic artery. The frontoethmoid suture is an important landmark during orbital surgery for approximating the floor of the anterior cranial fossa. The relation of the cribriform plate to the medial wall of orbit is variable. The vertical distance from the anterior cranial fossa to the medial canthal ligament at the level of the posterior lacrimal crest is between 0 to 19 mm. This distance may be

3mm or less in 20% of the individuals.[49] This should be kept in mind during dacryocystorhinostomy.

FLOOR OF THE ORBIT: Formed by

- Palatine bone
- Orbital plate of the maxillary bone
- Zygomatic bone

The floor of the orbit is situated above the maxillary sinus. The infraorbital groove which runs through the orbital aspect of the maxillary bone extends anteriorly from the inferior orbital fissure which transmits infraorbital vessels and the infraorbital nerve, a branch of the maxillary division of the trigeminal nerve. Fractures of the orbit most commonly involve the portion of the orbital floor which lies medial to the infraorbital groove. Floor is the shortest of the orbital walls. It is shaped like an equilateral triangle. The floor does not continue to the orbital apex. It ends 35 to 40 mm posterior to the rim at pterygopalatine fossa. This should be kept in mind during surgery on the floor because more posterior dissection may lead to extensive haemorrhage from the maxillary artery located in the pterygopalatine fossa.

The orbital floor remains thin medially with the maxillary sinus expansion but becomes strong lateral to the infraorbital nerve. The floor usually fractures at the site of unsupported dome of the maxillary sinus. In static loading studies, orbital floor shows the greatest degree of deformation.[43] This location is one of the convenient site for entry into the maxillary sinus during the orbital decompression surgery.

LATERAL WALL OF THE ORBIT: Formed by

- Zygomatic bone
- Greater wing of the sphenoid bone

The lateral orbital wall is the thickest of the orbital walls and separates orbit from the muscular temporal fossa anteriorly and middle cranial fossa posteriorly. At the apex of the orbit the sphenoid bone forms the lateral aspect of the superior and inferior orbital fissures. The spina recti lateralis lies at the junction of the wide and narrow portions of the superior orbital fissure on the surface of the sphenoid bone. The lateral rectus muscle arises from this bony projection.The zygomatic foramen which transmits the zygomatic nerve and vessels lies near the orbital rim of the zygomatic bone.

The vertical zygomatico sphenoid suture is the thinnest part of the lateral orbital wall. It forms a breaking point for bone removal during lateral orbitotomy. Temporalis muscle lies posterior and lateral to the lateral rim. The muscle has dense superficial fascia which can be easily harvested through skin and superficial muscle plane incision. The lateral orbital tubercle of theWhitnall[87] lies in the lateral orbital margin about 11 mm below the frontozygomatic suture. The lateral canthal ligament, check ligament of lateral rectus, lateral horn of the levator muscle, suspensory ligament of Lockwood and orbital septum attaches to this ligament. The zygomatico temporal and zygomatico facial foramina which transmits the neurovascular bundles perforate the anterolateral orbital wall.

APERTURES OF THE ORBIT:

OPTIC CANAL:

The optic canal is formed by the roots of lesser wing of the sphenoid bone is situated at the posterior end of medial wall of the orbit. The canal forms an angle of 36° to the midsagittal plane and is directed forward and laterally. The optic nerve with its meninges as well as ophthalmic artery which is embedded in the dural sheath of optic nerve pass through the optic canal. The artery lies inferior to the nerve for few millimetres and crosses the nerve to lie laterally after entering the orbit. The dimensions of the optic canals are symmetric measuring 4 to 4.5mm horizontally and6 to 6.5mm vertically.

SUPERIOR ORBITAL FISSSURE:

The superior orbital fissure is situated lateral to the optic foramen at the orbital apex. It is bounded by the greater and lesser wings of the sphenoid bone. The superolateral aspect of the fissure is narrower than the inferomedial part. At the junction of these two parts lies the spina recti. The annulus of zinn surrounds both superior orbital fissure and optic canal. The structures passing within the annulus of Zinn thus within the muscle cone are the optic nerve, the ophthalmic artery, superior and inferior divisions of the third nerve, nasociliary nerve , abducens nerve, and the sympathetic root of the trigeminal ganglion. The superior branch of inferior ophthalmic vein lies in the lower and medial compartment. The trochlear nerve, lacrimal and frontal nerves and the superior ophthalmic vein lies outside the annulus in the lateral aspect of the superior orbital fissure.

INFERIOR ORBITAL FISSURE :

The inferior orbital fissure is bound medially by the maxilla and the orbital process of the palatine bone and laterally by the greater wing of the sphenoid bone. It lies between the floor and medial wall of the orbit. Its anterior aspect is closed by the zygomatic bone. The venous drainage from the inferior orbit to the pterygoid plexus is transmitted through inferior orbital fissure. It contains neural branches from the pterygopalatine ganglion, infraorbital nerve and the zygomatic nerve.

ETHMOID FORAMINA:

The ethmoidal foramina in the medial orbital wall transmitsthe anterior and posterior ethmoidal arteries. It passes through the frontoethmoidal suture. This foramina may provide the route of entry into the orbit for infection and neoplasms from the sinuses.

The bony orbit contains fibroadipose tissues, extraocular muscles, cranial nerves supplying the eye and blood vessels. The fibroadipose tissue contains a connective tissue ‘ligament’ system described by Koornneef [47,48].The fibrous tissue extends from the periorbita to the tenon’s capsule and the fascial sheaths of the extraocular muscles and check ligaments. The orbital structures are supported by the connective tissue which is important for ocular motility. If this connective tissue is damaged by trauma restriction of extraocular movements occur.

The orbit also supports the eyelids. The orbital septum which arises from the arcus marginalis forms the intermediate connective tissue lamellae of the eyelids.[60] The eyelids are supported medially and laterally by medial and lateral canthal tendons respectively which is firmly attached to the orbital rim.[32,44] The medial canthal tendon is attached to the anterior lacrimal crest by anterior crus and to the posterior lacrimal crest by posterior crus. The eyelid globe apposition is provided by the posterior attachment.[44] The lateral canthal tendon is attached to the lateral tubercle of the zygoma inside the lateral orbital rim.[32] Injuries to the naso-ethmoid orbital complex can cause telecanthus and zygomatico orbital fractures can cause dystopia of lateral canthus.

SOFT TISSUES OF THE ORBIT:

PERIORBITA:

Periorbita lines the surface of the orbital bones. This tissue is loosely adherent to the bones. It is firmly fixed at the orbital margin, superior and inferior orbital fissures, lacrimal gland, optic canal and suture lines. The periorbita at the level of the optic foramen gives rise to the extraocular muscles. It gives fine processes into the orbit which divides the orbital fat into lobules and also supplies covering for nerves and vessels of the orbit. The periorbita becomes continuous with the endosteal layer of the dura at the level of the optic canal and superior orbital fissure. The periorbita becomes continuous with the periosteum of facial bones at the level of the margin of the orbit.

EXTRAOCULAR MUSCLES:

There are six extraocular muscles in the orbital cavity. The recti muscles pull the eye back into the orbit and the two oblique muscles pull the eye away from the orbit.[68] The four recti muscles arise at the level of orbital apex from the annulus of Zinn. The tendinous ring is made of two bands. The upper band arises from the body of the sphenoid and the lower band arises from the lesser wing of the sphenoid bone.

MEDIAL RECTUS MUSCLE

It is 40.8 mm long at its origin and is inserted 5.5 mm posterior to the limbus. The primary action of medial rectus is adduction. It is supplied by the inferior division of the oculomotor nerve. The blood supply is by medial muscular branch of the ophthalmic artery.

LATERAL RECTUS MUSCLE

It is 40.5 mm long at its origin and its tendinous termination is 9 mm long. It arises from the upper and lower parts of common tendinous ring. It is inserted 7 mm posterior to the limbus. It bridges the superior orbital fissure. The primary action of the lateral rectus is abduction. It is supplied by the sixth cranial nerve. The blood supply is by lacrimal artery and muscular branch of ophthalmic artery.

SUPERIOR RECTUS MUSCLE

It originates below the origin of levator palpebrae superioris muscle at the level of the common tendinous ring. It is 42 mm long and inserts in the sclera at about 7.7 mm posterior to the limbus. It is 6mm long at the level of its tendinous insertion. It forms an angle of 23° to the optical axis of the globe in its primary position. The action of superior rectus is elevation, intorsion and adduction. It is supplied by the superior division of the third nerve. The blood supply is from lateral muscular branch of the ophthalmic artery.

INFERIOR RECTUS MUSCLE

It is 40 mm long at its origin and is 5mm long at its tendinous insertion. It inserts 6.5 mm posterior to the limbus . It forms an ange of 23° to the optical axis of the globe in its primary position. The primary action of the inferior rectus is depression, secondary action being extorsion and tertiary action being adduction. It is supplied by the inferior division of the oculomotor nerve. Its blood supply is by medial muscular branch of the ophthalmic artery.

SUPERIOR OBLIQUE MUSCLE

It is the longest muscle of the orbit. It originates from the apex of the orbit medial to the optic foramen. It forms a cartillaginous pulley at the level of the trochlea which is attached inside the nasal orbital margin. It becomes tendinous at one centimetre before reaching the trochlea and passes posteriorly, inferiorly and laterally through the trochlea. It inserts in posterosuperior quadrant of the globe. The primary action is intorsion, depression being secondary action and abduction is the tertiary action. It is supplied by trochlear nerve. It get its blood supply from lateral muscular branch of ophthalmic artery.

INFERIOR OBLIQUE MUSCLE

It originates from the anterior part of the orbit below and lateral to the lacrimal sac. It passes backwards and laterally and is inserted into the lateral aspect of the globe. It has no tendon. The primary action is extorsion, the secondary action being elevation and the tertiary action is abduction.

CLASSIFICATIONOF ORBITAL FRACTURES

A) DEPENDING ON THE FRACTURED BONES

1) PURE OR INDIRECT BLOWOUT FRACTURE:

Fracture involving the orbital floor without associated rim or midfacial fracture.

2) IMPURE OR DIRECT BLOWOUT FRACTURE:

Assigned to cases with fracture of orbital wall associated with orbital rim and adjacent facial bones.

B) DEPENDING ON THE EXTENT OF FRACTURE:

a) Egg shell blowout fracture with hammock like sagging of floor into maxillary antrum.
b) Trapdoor blowout fracture: In this fracture, either edge of the inferior orbital wall is attached to its original position.
c) Linear blowout fracture capable of pinching the undersurface of orbital content.
d) Blowout fracture with large opening into the maxillary sinus

C) DEPENDING UPON THE LOCATION OF FRACTURE:

1) Diffuse (generalised) blowout fracture :Major portion of floor is collapsed into maxillary sinus.

2) Localised (limited) blowout fracture : It may be

   a) Medial blowout fracture usually occurs in impure type may be associated with nasoorbital fracture or fracture of medial orbital wall.

   b) Central blowout fracture usuallyof pure blowout type.

   c) Lateral blowout fracture is commonly of the impure type associated with zygomatic fracture.

D) DEPENDING ON THE DISPLACEMENT OF BONES:

a) BLOWOUT FRACTURE: Outward displacement of fractured bones resulting in net increase in intraorbital volume which causes enophthalmos.

b) BLOW IN FRACTURE: Inward displacement of fractured bones typically occurs at orbital roof resulting in relative reduction in orbital volume which causes exophthalmos. It occurs less commonly.[38,39,45]

E)DEPENDING ON THE PATTERN OF COMMINUTION:

Manson has devised a classification based on the pattern of comminution and displacement related to energy.[54]

a) Low-energy orbital fractures: Simple linear or circular blow-out fractures usually involving one or two walls. It typically involvs the floor or medial wall without involvement of orbital rim.

b) Middle energy orbital fractures: Involve at least two orbital wallsaccompanied by fracture of orbital rim. They do not involve the major posterior portion of the orbit.

c) High energy orbital fractures: Extreme disruption of multiple segments of the orbital rim with orbital walls. Usually they are circumferential, with three or four walls of the internal orbit are destroyed.

F) LE FORT FRACTURES:

a) Le Fort I: Low, transverse maxillary fractures which do not involve the orbit

b) Le Fort II: Fractures involving the nasoethmoid and maxillary bones with disruption of inferior and medial orbital walls.

c) Le Fort III: Fractures which extends from nasoethmoid region across the orbit, involving the the medial and lateral walls and the floor. Le Fort III facial fractures can cause craniofacial dysjunction if bilateral and complete in which facial skeleton is disarticulated from the skull.[66]

G) DEPENDING ON THE SOFT TISSUE INVOLVEMENT

a) CLOSED FRACTURE

Fracture associated with intact soft tissue component.

b) OPEN OR COMPOUND TYPE

Fracture communicates with the overlying skin.

MECHANISMS OF INJURY

The mechanism of blow out fracture of the orbit can be explained by two theories which explains why fracture of orbital walls occur without involvement of orbital rim.[29,35,71,84]

1) **Hydraulic theory:**

   Hydraulic theory was first explained by Smith and Regan in 1957.[84] Blowout fracture of the orbit results from sudden increase in intraorbital pressure due to trauma by a blunt object of size being greater in diameter than the orbital entrance. The orbital contents are compressed posteriorly towards the apex of the orbit. The posterior orbit cannot accommodate this increased volume of tissue, so the orbital bones break at their weakest point which is the posteromedial part of floor in maxillary bone. Also an associated blowout fracture of the thin medial orbital wall may occur at the same time and also by the same mechanism. The posteromedial aspect of orbit is involved in 75% of blowout fractures.

2) **Buckling force theory (by Fujin) :**

This theory suggests that the striking object can causea compressive force on the inferior orbital rim which leads directly to the buckling of the orbital rim. The bony orbital floor is fractured directly by this buckling force. This also explains why it is possible for a blow in fracture of the orbit to occur in which the orbital floor is fractured into the orbit but not into the maxillary sinus. The degree of increased intraorbital pressure determines whether orbital tissue are pushed down through the fracture into the maxillary antrum or not.

Koornnoef described about the fine connective tissue septa of the orbit that envelope the muscle, globe and the intraorbital contents and also periorbita of the orbit. In the case of an orbital fracture, these connective tissue septa, rather than the extraocular muscles themselves may become involved in the fractured site. Because of the delicate interconnection of these septa with local and distant structure on the orbit , bizzare motility problems can develop following an orbital wall fracture without a significant amount of incarcerated tissue being visible by x ray.

CLINICAL FEATURES

Clinical features vary according to the severity of trauma and the time interval between injury and presentation of patient. Therefore evaluation of a patient with orbital fracture should be proceeded in a logical and defined manner.[22,67,76]

Initially there will be periorbital edema and extravasation of blood in and around the orbit.

Emphysema of the eyelid may occur more frequently following fracture of medial wall than that of the floor which may be made worse by blowing of the nose. Crepitus, a crackling sound on palpation is a sign of air entrapment in soft tissues.

Ipsilateral epistaxis can occur as a result of bleeding from maxillary sinus into nose is an early sign. They may present with variable degree of proptosis initially because of the associated orbital emphysema, edema and haemorrhage.

ENOPHTHALMOS:

Usually occurs 10 days after the trauma due to resolution of the causes of proptosis, the eyeball is displaced backwards and downwards causing enophthalmos.

Factors responsible for producing enophthalmos are

1) Enlargement of orbital cavity from displacement of the fractured fragments
2) Escape of orbital fat into maxillary sinus
3) Backward traction of globe by entrapped inferior rectus
4) Necrosis of orbital fat may result from low grade inflammation or by the pressure from an orbital haemorrhage

DIPLOPIA

Diplopia is a common and important symptom usually due to restriction of vertical movements in some cases it may become the dominant permanent disability. It typically occurs in both up and downgaze (double diplopia). Main factors responsible are

1) Incarceration of soft tissue within the fracture site particularly the inferior rectus and inferior oblique muscles, causing

displacement of ligament of lockwood, periorbita, muscle sheaths and their connection.

2) Direct injury to muscle

   - through laceration of bony fragments

   - haemorrhage into their substance

3) Damage to the nerve supply to the muscle occur either as they enter the muscle or at the orbital apex may resulting in a paralytic squint.

4) Diplopia may also occurs due to the secondary deviation due to overaction of the conjugate muscle of the opposite eye.

Muscle traction test is useful in the detection of the incarceration of a muscle as distinct from the limitation of movement due to swelling of the soft tissue or haemorrhage.

Following combination of diplopia and enophthalmos may be observed in blowout fracture.

1) Diplopia with enophthalmos –results from incarceration of orbital content in the area of the fracture.

2) Diplopia without enophthalmos – occur with fixation of orbital contents in a linear fracture. There is no enophthalmos

because there is no escape of orbital fat and no enlargement of orbit.

3) Enophthalmos without diplopia – occur when the inferior orbital contents are not entrapped in the fracture. The periorbita is torn and an opening is created allowing escape of orbital fat only. Otherwise the orbital cavity may be sufficiently enlarged to cause enophthalmos.

4) No diplopia no enophthalmos –occurs when the fracture neither causes fixation of the orbital content nor disturbs the anatomy of periorbital or orbital cavity.

Forced duction test

It is performed with instillation of local anaesthetic in the cul de sac. The insertion of the inferior rectus muscle is grasped through the conjunctiva with toothed forceps and the globe is rotated up and down. If inferior rectus entrapment is present, there will be passive restriction of the globe on upgaze suggestive of inferior rectus entrapment. Also comparing the intraocular pressure in primary position and in upgaze suggests inferior rectus entrapment if there is significant increase in intraocular pressure in the upgaze.

SENSORY LOSS(PARESTHESIA AND ANAESTHESIA)

An important sign is anaesthesia in the infraorbital area viz

- lower lid
- cheek
- side of nose
- upperlip
- upper teeth
- oral mucosa

supplied by infraorbital nerve although this may be difficult to elicit. Infraorbital anaesthesia suggests a fracture in the central part of the floor of the orbit. The absence of such sensory loss associated with other signs of fracture of orbital floor indicates that the injury may be either medial or lateral to the infraorbital canal.

There is no associated severe ocular damage in a blowout fracture because blowout fracture itself is a protective mechanism for globe. The eye should be carefully examined to exclude the possibility of intraocular damage.

This includes

- hyphema

-traumatic iridodialysis

-contusion cataract

-lens subluxation

-angle recession

-vitreous haemorrhage

-retinal dialysis

-giant retinal tear

-commotio retinae

-preretinal haemorrhage

WHITE EYED BLOWOUT FRACTURE

It is common in paediatric patients. There will be signs of entrapment of extraocular muscles with a relatively white eye suggestive of minimal signs of inflammation. They also have symptoms suggestive of oculocardiac reflex with resultant bradycardia and nausea.

ORBITAL ROOF FRACTURE

Orbital roof fractures caused by blunt trauma is more common in young children, because the frontal sinus is not pneumatised. In older patients, the force absorbed by frontal sinus which prevent injury to the roof. Serious complications which can arise are cerebrospinal fluid rhinorrhea, intracranial injuries, pneumocephalus, and subperiosteal hematoma. Entrapment of extraocular muscles is rare, and diplopia if it occurs may be due to edema, hematoma or contusion of orbital structures. Most of the roof fractures do not require surgical intervention.

Fracture of medial orbital wall

Direct fractures

They commonly involve ethmoid bone, frontal process of maxilla and the lacrimal bone. They may present clinically with depressed bridge of the nose and telecanthus. They may be divided into the following types.

Type I- Central fragment of the bone attached to the canthal tendon.

Type II- Communited fracture of the fragments in the centre

Type III- Avulsion of the tendon or communition of the tendon attachment.

Complications like epistaxis due to rupture of anterior ethmoidal arteries, cerebral damage, cerebrospinal fluid rhinorrhea, lateral displacement of the medial canthus and damage to the lacrimal drainage system can occur.

Indirect fractures occurs due to extension of fracture of the orbital floor. Surgical intervention is unnecessary in cases of medial blowout fractures unless the medial rectus muscle is entrapped.

ORBITAL FLOOR FRACTURE

Orbital blowout fracture should be suspected in a case of blunt injury to the orbital wall in which the periorbital blow is forceful enough to cause ecchymosis. They may have diplopia of upgaze or downgaze, enophthalmos, ptosis and emphysema of the orbit and the lids.

COMPARTMENT SYNDROME

Visual loss in orbital fractures can occur due to injury to the optic nerve or increased orbital pressure causing compartment syndrome. Increased intraorbital pressure can cause central retinal artery occlusion or occlusion of the posterior ciliary arteries. Therefore any patients with tight orbit, decreased vision with increased intraocular pressure should undergo emergent orbital decompression. Lateral canthotomy with or without inferior cantholysis can be done.

COMPLICATIONS OF ORBITAL FRACTURE

1) Immediate complications

- Loss of vision – nerve/ vascular/ globe injury
- Pulsating exophthalmos
- Orbital and periorbital bleeding, epistaxis
- Orbital emphysema
- Skeletal and structural derangement
- Soft tissue and muscle entrapment or derangement

2) Delayed complications

- Nasoorbital skeletal disruption
- canthal disruption or derangement
- Lacrimal drainage problems
- Orbital, periorbital structural derangement
- Exorbitism secondary to lateral wall fracture

- Enophthalmos secondary to volume changes in the orbit

- Vertical diplopia

3) Late complications

- Extraocular muscle imbalance

- Enophthalmos

- Misplaced orbital floor

- Eyelid problems

- Reconstruction failures

EVALUATION

Following is the overview of the initial examination of a patient with orbital trauma.

ASSESMENT OF A PATIENT

History

- Mechanism of injury
- Loss of consciousness

Systemic assessment (in acute cases)

- Vital signs
- Quick systemic review

Ocular assessment

- Visual acuity
- Pupil
- Anterior segment
- Intraocular pressure
- Dilated fundoscopy

Orbital assessment

- Face, eyelids and adnexa
- Globe position and exophthalmometry

- Orbital margin and emphysema

Ocular motility evaluation

Motor abnormalities

- Ocular misalignment
- Limited range of motion
- Globe retraction
- Alteration of palpebral fissure width
- Nystagmoid movements

Sensory abnormalities

- Diplopia
- Visual confusion
- Loss of stereopsis
- Limitation in the field of view

Evaluation of above abnormalities needs the following

- Binocular eye movements (Hess charting)
- Diplopia charting
- Quantitative evaluation of strabismus with prism
- Traction testing
- Tests of binocular function and colour vision

Sensorimotor examination

- Loss of sensation / numbness of lower lid and cheek

- Numbness of upper teeth and mucous membrane of upper lip

- Numbness around upper lid and upper eyebrow

- Evaluation of orbicularis, frontalis and levator muscles

Nasal examination

- Inspection and palpation of nose

- CSF rhinorrhoea

Radiological evaluation

- Clinical evaluation must be correlated by roentgenographic studies. Water's view is set in the nose chin position to view the orbital floor, zygomatic bones, sphenoid ridges and temporal arches.

- Coronal CT scans accurately predicts the relationship between orbital bone, muscle and soft tissue.

X rays:

Water's view is used for the detection of orbital fractures. The other views which are used are Caldwell, frontooccipital projection, oblique view and anteroposterior view.

The common findings are

1) Fragmentation and

2) Depression of bony fragments

3) A ‘tear drop’ opacity of superior maxillary antrum from orbital contents herniating through the floor.

In addition, the following finding may be seen.

1) Increased orbital density associated with thickening of soft tissue shadow over inferior orbital rim due to edema.

2) Fracture of inferior orbital rim

3) Partial to complete opacification of underlying maxillary sinus associated with haemorrhage.

4) Orbital emphysema which occurs with associated ethmoid fracture.

5) Fracture medial orbital wall which is indicated by extension of orbital soft tissue shadow into ethmoid cells.

Not all blowout fracture are seen radiologically. The ‘trapped door’ variety has minimum bony displacement and floor deficiency. Radiographic diagnosis is a matter of considerable importance. The radiological technique must be above the average standard to consider its usefulness. The bony walls in this region are thin and are often obscured by thicker bones while the fracture lines which are readily confused with the suture lines and bony septa in this area may at one time appear as rarefaction in the film and at another as lines of increased density where the fragments overlap. In majority of cases thereafter ordinary direct posteroanterior and lateral views of the skull are insufficient and stereoscopic and other special views are required to demonstrate the profile of the orbital floor, supplemented on occasion by the techniques of tomography.

MANAGEMENT OF ORBITAL FRACTURES

The primary consideration in the management of orbital fractures is to determine which fractures require surgery and when such intervention should be undertaken.

Conservative management is indicated in case of

1) Minimal diplopia with good ocular motility with evidence of clinical improvement
2) CT scan showing no evidence of muscle entrapment.
3) Absence of significant enophthalmos or hypo-ophthalmos & small orbital wall fractures unlikely to produce late enophthalmos.

Surgical intervention is needed in cases of

1) Clinically significant diplopia with positive forced duction test or radiological evidence of muscle entrapment showing no improvement over 1 to 2 weeks.
2) Early enophthalmos of atleast 3 mm or significant hypo-ophthalmos.
3) Large fractures involving atleast half of the orbital floor.
4) Associated displaced orbital rim or facial fracture.

Timing of repair :

Previously it was considered that orbital fractures should be repaired within 48 hours of trauma to get a satisfactory result. It is now established that the optimal time for surgery when indicated is 10 to 14 days of injury. During this period local edema and periorbital hematoma will settle sufficiently to allow adequate examination of ocular position and movements. However, late primary treatment beyond 21 days after malunion of fractures and fibrosis of ocular structures is more difficult.

Orbital implants :

The orbital implants include allogenic grafts and alloplastic materials. The autogenous implants used are bone grafts, cartilage and fascia. Among these bone graft is commonly used. They have excellent biocompatibility but require a second procedure to harvest a graft with attended donor site morbidity and increased operating time. The sites for harvesting bone grafts include iliac crest, cranium and rib. Cranial bone is favoured by many because it is a membranous bone and undergoes less resorption than enchondral bone(rib, iliac crest). The anterior wall of maxillary antrum is also a source of membranous bone but the amount of bone available for grafting is limited to about 1x1.5 cm in adults.

The alloplastic implants available for orbital reconstruction can be categorised as porous, non porous and absorbable. Non porous implants include metallic implants usually composed of titanium or vitallium such as miniplates, microplates, grids and meshes. Additional non porous implants include Supramid, silicone, Gore-Tex and Teflon. The porous implants allows fibrous ingrowth which has the following advantages

1) It anchors the implant to surrounding orbital tissues rendering the implant less likely to migrate or extrude.
2) Able to resist late infection.

Commercially available porous implants include hydroxyapatite and high density porous polythelene. Hydroxyapatite is hard and brittle limiting its usefulness as orbital floor implant. Absorbable implants like Gelfilm and polygalactin are available for orbital fractures but they are generally limited to smaller fractures that do not require great deal of rigid support.

The advantages of alloplastic implants include decreased operating time, absence of early resorption, lack of donor morbidity and increased ease of handling. Complications associated with orbital implants depend not only on the type of implant but also on the surgical technique employed. Proper positioning and stable fixation of the alloplastic implants decreases the rate of complications.

Orbital floor fractures:

The orbital floor may be approached through eyelid, canine fossa or through maxillary sinus. Eyelid approach is usually referred as it allows easy disengagement of entrapped orbital tissue. Approach through maxillary sinus and canine fossa is indicated for comminuted fractures of maxilla.

The surgical approach to floor fracture can be made through infraciliary incision, or transconjunctival incision with or without lateral cantholysis and inferior lid crease incision. Currently the commonly preferred incision was 3 mm below the lash margin as it leaves minimal scar.

Forced duction test is done at the beginning of surgery to assess the degree of restriction. The incision is made through skin and orbicularis until tarsus is reached. Dissection is continued until rim of orbit is reached. Periorbita is elevated from the floor and prolapsed tissue is released from the fracture. If the fracture is small, linear or of trapdoor variety no implants are needed. Otherwise bonegrafts or alloplastic implants like porous polythelene, Supramid, Gore-Tex,Teflon,Silicone sheet or titanium mesh can be used to fill the gap.

Medial orbital fractures :

When dissecting the medial orbit, the surgeon should be careful to avoid damaging the lacrimal canaliculi and sac, medial canthal tendon, trochlea, superior and inferior oblique muscles. The commonly used surgical approaches are Lynch incision and transcarancular approach. The Lynch or frontoethmoid incision is made vertically just medial to the insertion of medial canthal tendon approximately 9-10 mm medial to the medial canthal angle. Once the incision is made blunt dissection is made and periosteum is incised. The lacrimal sac is identified and separated from the fossa. The fracture site is identified and periosteal elevator is used to remove the incarcerated tissue. Forced duction test is performed to ensure complete release of entrapped tissue. The bony defect is closed with autogenic grafts or alloplastic materials.

Orbital roof fractures :

Most of the roof fractures do not require repair. The indications for surgery are generally neurosurgical, and the treatment often involves team approach with neurosurgeon and orbital surgeon. The approaches are transcutaneous incision incision in the upper lid crease and trans conjunctival incision.

Lateral wall fracture :

The incision used for lateral wall fracture is S shaped Stallard Wright incision which extends from beneath the eyebrow laterally and curving down along the zygomatic arch allows good exposure of lateral rim but leave a noticeable scar.

It has been replaced by newer approaches through upper eyelid crease incision or lateral canthotomy incision.

POSTOPERATIVE COMPLICATIONS

Post operative complications of orbital fractures can be minimized by good surgical technique. However, because such injuries are frequently associated with soft tissue damage, post operative complications may still occur. Some of the complications are common to all the types of surgery, some specific to the malposition of orbital rims and others related to disruption of internal orbital walls.[59] The most serious complication of orbital surgery is visual loss. The intraoperative hemorrhage that produces compressive optic neuropathy or central retinal artery occlusion. Early detection of the visual loss is essential that is why occlusive dressings are avoided and the patient should be followed closely after surgery.

Pupillary examination to detect afferent pupillary defect is a reliable method to detect optic neuropathy. Orbital haemorrhage in such cases is obvious and when it is found to be causing visual impairment it can be treated by lateral canthotomy, cantholysis and adjunctive medical and surgical treatment.[76] Repeat orbital CT is appropriate in such cases.

Soft tissue disruption associated with orbital fractures produce late sequelae like inferior dystopia with displacement of lateral canthus and displacement of medial canthus producing telecanthus. Mild lateral canthal dystopia can be managed by lateral canthoplasty and repositioning of lateral canthus with tarsal strip procedure provided that the malar position / malar rim is not significantly displaced. Severe forms of diplopia necessitate osteotomy and repositioning of the displaced lateral orbital rim and zygoma. Persistent telecanthus is a frequent complication of nasoethmoid fracture. Treatment is performed with medial canthopexy using transnasal wiring or other fixation techniques. Excess scar tissue and displaced bony fragments should be removed from the medial canthal region to achieve a satisfactory result. Damage to the nasolacrimal drainage system may be a sequel of nasoethmoid or midfacial fracture. If injury to the nasolacrimal drainage system is obvious primary repair should be performed. In most of the cases, injury to the nasolacrimal system is not noted until later, manifesting as nasolacrimal duct obstruction producing epiphora with or without dacryocystitis. Treatment is by dacryocystorhinostomy.

The complications which can occur after repair of internal orbital fractures include persistent enophthalmos or hypo-ophthalmos and diplopia as well as those related to synthetic orbital implants or bone grafts. The potential complications related to the alloplastic implants and bone grafts include migration, extrusion and infection. Bone grafts have the complication of variable degrees of resorption which is more in enchondral bone compared with membranous bone grafts. Infection or extrusion usually requires removal of the implant or bone graft if improvement with antibiotic is not seen. The implant should not be replaced in the setting of infection. In many of the cases, fibrous tissue forms around the alloplastic implants after after several weeks or months, which allow enough orbital tissue support which does not require replacement implant. However, in other cases secondary repair with suitable alloplastic implant or graft must be performed after adequate control of infection. Observation should be done in cases of migration of alloplastic implant or bone graft if it is minor and not causing significant sequelae. Marked implant migration causing functional impairment necessitates removal of the bone graft or alloplastic implant.

The globe malpositions after blowout fracture repair are caused by failure of the surgery to reconstruct the normal anatomical boundaries of the orbit. The soft tissue scarring in a small number of cases may occur due to late enophthalmos. The enophthalmos may be corrected by repositioning the implant or by keeping alloplastic implant or bone graft to augment the volume deficiency. Camouflage techniques such as ptosis repair of the ipsilateral lid or blepharoplasty of the contralateral upperlid may also be done. Diplopia which occurs after blowout fracture repair may be due to extraocular muscle injury or neurological injury or due to persistent soft tissue entrapment. In the perioperative period there may be limitation of ocular motility due to postoperative edema, therefore ocular motility should be reassessed after the edema resolves which usually occurs in 2 to 3 weeks. Repeat CT scanning is also helpful. If the clinical examination and CT scan findings are suggestive of extraocular muscle entrapment in a patient with functionally significant diplopia, early orbital re-exploration is indicated.

The persistent ocular motility defects which are not amenable to orbital surgery usually requires strabismus surgery. A period of 6 months or more is allowed for spontaneous improvement. Diplopia may be treated with fogging, Fresnel prisms, or patching during this waiting period. Young children who are at risk of amblyopia require close followup. Repair of the blowout fracture is successful if the diplopia is relieved within the functional 30° fields of gaze. Reoperation is not indicated for diplopia occurring in more eccentric fields of the gaze, although each case must be individualised.

REVIEW OF LITERATURE

In the 19th century, Lang[50] provided the first clinical description of blowout fractures of orbit. He summarised the pathomechanisms in 1889. He suspected the orbital enlargement as the cause of enophthalmos which was reinforced by LaGrange.

In 1940 King and Samuel[46]elaborated on Lang's postulation and attributed transmission of the force through the globe to the orbital walls and abrupt increase of intraorbital pressure being the main factor implicated.

In 1943, Converse described the advantages of bone implantation to correct enophthalmos in orbital floor fracture as against cartilage which tends to get absorbed.

McGrigor and Samuel in 1945 first described the radiological diagnosis of blowout fractures of orbit.

Converse[11] and Smith in 1960 proposed that diplopia and vertical muscle imbalance was caused by herniation and entrapment of the inferior rectus and inferior oblique muscles into the defect caused by orbital fractures.

Cavol Browning[9,10] in 1965, first used the inert alloplastics in 45 cases of orbital floor reconstruction without implant related complications. They used materials like polyethylene, silicone DC, cranioplast and supramid.

Lerman[52] preferred the infraorbital approach as it avoids damage to the orbital septum.

Hotte[41] in 1970, described the rarity of actual muscle entrapment in blowout fractures and felt that incarcerated fat by virtue of its connections to the muscles tethered the muscle indirectly to the fracture site.

Miller and Glasser[62] in 1966, coined the term 'retraction syndrome' in a case of medial wall fracture with entrapment of medial rectus muscle and this was later followed by similar reports by Edward[24,26] and Rumult[78].

Melvin Butler[57] in 1971, favoured infraorbital approach as it relieved the diplopia persisting after antrostomy.

Richard Ziff[73] in 1973 reported a case of traumatically induced Brown's syndrome following a orbital floor fracture which mimics a superior oblique tendon sheath syndrome.

Fujino[29] in 1980, proposed that buckling forces transmitted to the floor causes most of the fracture due to direct blow to the orbital rim. The rim will remain intact but would bend resulting in reciprocal deformation of the floor.

Daniel Mc Lachlan[17] in 1983, reported visual loss[42], enophthalmos, extraocular muscle imbalance, pulsatile exophthalmos[74] and intracranial complications following orbital roof fractures.

Vinik Garano[85] reported that fracture medial part of the roof of the orbit may involve frontal sinus and fracture lateral part of the roof may involve supraorbital nerve and levator palpebrae superioris.

Godoy[34] in 1985, reported a case of enophthalmos with malar fracture.

Dingman[72], and Freeman[28] suggested early surgical management of zygoma fracture to prevent functional and cosmetic deformities.

Milman[63] in 1987, suggested steroid protocol consisting of 1 mg/kg of prednisolone to hasten the resolution of edema to unmask the patients whose diplopia will not resolve.

Patrich Yeatts[69] et al in 1992, suggested a modification of Hertel exophthalmometer with reference point as the external auditory meatus to measure the globe position in complex orbital fracture where erroneous results can occurred if measured from lateral orbital rim.

Bedisonian[3] in 1993, advocated the use of banked fascia lata in orbital floor fractures and Mauriello[a] advocated vicryl as an orbital floor implant.

Marco Nardi[55] in 1996, provided a mechanism for hypertropia in orbital blowout fracture. Marco Nardi suggested that due to dragging force of inferior rectus there is a postinflammatory displacement of inferior oblique owing the fusion of both in sheaths.

Rene'Le fort delineated the lines of weakness along which orbital fractures occurred and John Marquis Converse stressed about the fractures which occurs in the delicate walls of the orbit following less severe orbital trauma.

Murphy[64] was the first surgeon to use osteoperiostel graft in enophthalmic eye with floor fracture.

Devoe[19] was the first person to propose a non-operative approach in orbital fractures and found that small amount of enophthalmos and diplopia which were not functionally limiting will disappear.

Converse and Smith[12,16] described the implantation of bone in fracture of the orbital floor.

Callahan[6] described the use of titanium mesh which was later followed by the use of Teflon by Sounder. Still later methyl methacrylate and Supramid implants came into use.

David B Soll[18] described the trap door variety of orbital blowout fractures of the orbit. Converse and Smith described Compartment syndrome following a small defect which would trap the muscle causing marked limitation of the movements.

Michael Kroll[61] compared three different surgical approaches namely infraorbital approach, Caldwel Luc and combined approach[25] and found that infraorbital approach was the preferred approach the other approaches were used in case of inadequacy with infraorbital approach.

Converse[13] emphasised the benefits of early surgical repair in a 10 year survey of orbital fractures.

Converse[15] emphasised delayed surgical treatment for patients with residual symptoms. This causes incomplete correction of enophthalmos and diplopia. However opinion remain divided into medical[65,70] and surgical therapies[23,28].

Ronald Goldman[75] emphasised the use of Caldwel Luc procedure for primary repair.

AIMS AND OBJECTIVES

1) To analysis the causes of orbital fractures.

2) To determine the types of orbital fractures encountered.

3) To document the clinical presentation of orbital fractures and associated ocular injuries.

4) To study the management of orbital fractures.

INCLUSION CRITERIA

1) All cases of orbital fractures proven radiologically.
2) Patients with complaints of restriction of ocular movements with or without defective vision.
3) Patients with complaints of diplopia and enophthalmos.

EXCLUSION CRITERIA

1) Cases with ocular fibrosis.
2) Patients unwilling for radiological investigations.

MATERIALS AND METHODS

A retrospective study of 56 cases of orbital fractures was conducted at Madurai medical college, Madurai over a period of 3 years. All cases with CT scan or radiological evidence of orbital fractures were included in this study irrespective of age and sex and the following parameters were recorded in each case.

1) Age
2) Sex
3) Eye involved (laterality)
4) Cause of injury
5) History of epistaxis
6) Ocular examination

a) Visual acuity
b) Anterior segment evaluation
c) Fundus examination
d) Examination of lids
e) Examination of orbital margins for irregularity, gaps, tenderness and emphysema.

f) Ocular motility – elevation,depression, abduction and adduction were quantified as mild, moderate and severe restriction.
g) Infraorbital anaesthesia
h) Axial globe displacement (exophthalmos, enophthalmos) was measured by Hertel's exophthalmometry.
i) Diplopia charting was done in all cases wherever applicable.
j) Intraocular pressure by Schiotz tonometry.
k) Force duction was done in all possible cases with ocular movement restriction.

INVESTIGATION

All cases had CT scan or Water's view xrays during their first visit. The findings were documented as follows.

a) Walls involved
b) Rim involvement
c) Entrapment of soft tissues
d) Entrapment of extraocular muscles
e) Maxillary sinus involvement
f) Orbital emphysema

TREATMENT

Inj. Tetanus Toxoid 1 ampoule intramuscularly was given for patients with skin lacerations.

a) Medical management

1) Patients with no functionally limiting diplopia or resolving diplopia, negative force duction tests, cosmetically acceptable enophthalmos and or no entrapment of muscle or soft tissue on CT scan were managed medically.
2) Medical management consists of observation undercover of systemic antibiotics and anti-inflammatory drugs.

b) SURGICAL MANAGEMENT

Indications for surgical treatment included:

-Functionally limiting diplopia in primary and or downgaze not resolving within 2 weeks of observation.

- Positive forced duction test.
- Significant enophthalmos of 2 mm or more.
- CT scan evidence of a large fracture and or muscle entrapment.

Surgery was done under general anaesthesia. Depending on the location and type of fracture and age of the patient appropriate approach was chosen. Most cases of floor fracture were approached infraorbitally and few transconjunctivally. Patients with fractures involving inferior and medial walls were approached through inferomedial incision. Stallard wright incision and or upper brow incision were used in cases with lateral wal fractures.   Following reduction of fracture implants prolene mesh or titanium mesh were used in appropriate cases. Plate fixation of lateral wall fracture was done in one case. Postoperatively patients were managed with the systemic antibiotics and anti-inflammatory drugs. Suture removal was done between 5 to 7 days.

RESULTS

DEMOGRAPHICS

TABLE 1

Age (Yrs)	Male	Female	Total	Percent (%)
<=10	4		4	7.1
11-20	10		10	17.8
21-30	18	4	22	39.2
31-40	12		12	21.4
41-50	6	1	7	12.5
51-60	1		1	1.7
Total	51	5	56	99.8

Orbital fractures frequently involved in the age group of 21 to 30 years (39.2%).

Table 2

Laterality

Laterality	Patients	Percent (%)
Right eye	32	57.1
Left eye	24	42.8
Both eyes	0	0
Total	56	

57.1% of patients had involvement of their right eye and 42.8% of the patients had their left eye involvement.

Table 3

Causes of orbital fractures

Activities	Male	Female	Total
Road Traffic accidents	20		20
Sports/ Recreation	8		8
Violence	12	3	15
Domestic	2	2	4
Falls	5		5
Occupation	4		4
Total	51	5	56

Most of the orbital fractures were due to road traffic accidents.

Table 4

CLASSIFICATION OF FRACTURES

Classification	Eyes	Percent (%)
A) Pure blowout	23	41
Inferior	15	26.7
Medial	5	8.9
Medial Inferior	3	5.3
B) Impure blowout	25	44.6
Inferior	10	17.8
Medial	5	8.9
Inferior medial	10	17.8
C) Other fractures	8	14.2
Total	56	

41% of patients had pure blowout fractures and 44.6% of patients had impure blowout fractures.

Table 5

Sites of orbital fractures

	Eyes	Percent (%)
A) Isolated fracture		
Inferior	25	44.6
Medial	10	17.8
Superior	3	5.3
Lateral	3	5.3
B) 2 wall combination		
Inferior + Medial	13	23.21
Inferior + Lateral	1	1.7
Superior + Lateral		
Superior + Inferior		
Superior + Medial		
Medial + Lateral		
A) 3 wall combination		
Floor, Medial & Lateral	1	1.7
Floor, Roof & Lateral		
Total	56	

The inferior wall was most commonly fractured in 44.6% of patients and medial wall in 17.8% of patients.

TABLE 6

CLINICAL PRESENTATION OF ORBITAL FRACTURES

CLINICAL FEATURES	EYES	PERCENT
Periorbital ecchymosis	50	89.2
Extraocular movement restriction	42	75
Diplopia	26	46.4
Hypotropia	6	10.7
Hypertropia	10	17.8
Infraorbital anaesthesia	25	44.6
Enophthalmos	22	39.2
Emphysema	7	12.5
Epistaxis	8	14.2
Ptosis	6	10.7

89.2% of patients in my study group has periorbital ecchymosis and 75% of patients has extraocular movement restriction.

TABLE7

OCULAR INJURIES ASSOCIATED WITH ORBITAL FRACTURES

OCULAR INJURIES	EYES	PERCENT
Hyphema	3	5.3
Iridocyclitis	5	8.9
Iridodialysis	4	7.1
Subluxation of lens	2	3.5
Dislocation of lens	1	1.7
Traumatic cataract	5	8.9
Optic neuropathy	3	5.3
Macular edema	3	5.3
Retinal detachment	1	1.7
Choroidal rupture	3	5.3
Vitreous haemorrhage	2	3.5
Globe rupture	2	3.5

Hyphema, iridocyclitis, iridodialysis, subluxation and dislocation of lens, traumatic cataract, optic neuropathy, macular edema, vitreous haemorrhage and globe rupture were most common ocular injuries associated with orbital fractures.

Table 8

BASELINE OCULAR DEVIATION

	Eyes	Percent (%)
Primary	32	57.1
Hypertropia	10	17.8
Hypotropia	6	10.7
Exotropia	6	10.7
Hypo & exotropia	2	3.57
Total	56	

17.8% of patients had hypertropia and 10.7% of patients had hypotropia.

Table 9

EXTRAOCULAR TISSUE ENTRAPMENT

	Eyes	Percent (%)
Soft tissue entrapment	26	46.4
Muscle entrapment		
Inferior rectus	16	28.5
Medial rectus	8	14.2
Superior rectus	2	3.5
Inferior & medial rectus	2	3.5
Inferior rectus & inferior oblique	2	3.5
None	26	46.4

46.4% of patients had soft tissue entrapment, 28.4% of patients had inferior rectus entrapment and 14.2% of patients had medial rectus entrapment.

Table 10

SINUS INVOLVEMENT

	Eyes	Percent (%)
Maxillary sinus		
Haziness	24	42.8
Fluid level	8	14.2
Tear drop	6	10.7
None	18	32.1
Ethmoid sinus		
Haziness	8	14.2
Fluid level	8	14.2
None	40	71.4

42.8% of patients had maxillary sinus haziness, 14.2% of patients had ethmoid sinus haziness and 10.7% of patients had tear drop sign.

Table 11

BASELINE FORCED DUCTION TEST

FDT	Eyes	Percent (%)
Elevation	18	32.1
Abduction	6	10.7
Others	5	8.9
Negative	25	44.2
Missing	2	35
Total	56	

32.1% of patients had restriction of elevation by forced duction test and 10.7% of patients had restriction of abduction.

Table 12

ASSOCIATION BETWEEN WALL INVOLVEMENT AND ENOPHTHALMOS

	Enophthalmos	Exophthalmos
Floor	10	1
Floor & Medial wall	5	2
Floor, Roof & Lateral wall	2	0
Floor, Medial & Lateral	1	0
Floor & Roof	0	1
Medial wall	2	1
Roof	1	0
Lateral wall	1	0
Total	22	5

28 eyes were normal and in 1 eye Hertel could not be determined.

Table 13

BASELINE VISUAL ACUITY

	Eyes	Percent (%)
No PL	3	5.3
PL	2	3.5
1/60	2	3.5
3/60	2	3.5
5/60	2	3.5
6/60	2	3.5
6/12	10	17.8
6/9	7	12.5
6/6	26	46.2

46.2% of patients had visual acuity of 6/6 and 5.3% of patients had visual loss.

Table 14

TYPES OF FRACTURES BY TREATMENT

Types of fracture	Surgical	Medical	Total
Pure blow out	11	12	23
Impure blow out	5	20	25
Lateral Wall	1	2	3
Others	-	5	5
Total	17	39	56

39 patients underwent conservative management and 17 patients underwent surgical correction.

Table 15

TREATMENT

	Eyes	Percent (%)
Medical	39	69.6
Infraorbital	11	19.6
Medial orbital	2	3.5
Transconjunctival	2	3.5
Transconjuctival & Medial orbital	1	1.7
Others	1	1.7
Total	56	

In our study, 69.6% of patients underwent conservative management and 30.3% of patients undergone surgical correction. Among the surgically treated cases, the most common approach was through infraorbital incision (19.6%).

Table 16

IMPLANTS USED

Implants	Floor	Medial wall	Floor & Medial wall	Roof	Lateral wall
Prolenemesh	4	3	2		
Titanium mesh	1		1		
Plate fixation	1				1
No implants	2	1	1		
Total	8	4	4		1

Prolene mesh was most commonly used for repair of blowout fracture of orbit. Lateral wall fracture was treated by plate fixation.

Table 17

CHANGE IN EXOPHTHALMOMETRY

Difference in mm	Baseline (%)	Final (%)
-3	5(8.9%)	0(0%)
-2	13(23.2%)	9(16%)
-1	4(7.1%)	15(26.7%)
0	22(39.2%)	29(51.7%)
+1	6(10.7%)	1(1.7%)
+2	4(7.1%)	1(1.7%)
Not recorded	2(3.5%)	1(1.7%)
Total	56	

This table shows that significant enophthalmos >2mm has decreased from 32% at baseline to 16% in final followup.

Table 18

COMPARISON OF BASELINE AND FINAL OCULAR DEVIATION

Ocular deviation	Baseline (%)	Final (%)
Primary	57.1	70
Hypertropia	17.8	10
Hypotropia	10.7	10
Exotropia	10.7	7
Hypo & Exotropia	3.57	3
Total	99.87	100

In this table, hypertropia and exotropia have decreased significantly in the post treatment period.

Table 19

COMPARISON OF BASELINE AND FINAL FORCE DUCTION TEST

Test Result	Baseline (%)	Final (%)
Positive	47	19
Negative	49	77
Not recorded	4	4
Total	100	100

This table shows that there was a reduction in the positive force duction test in the final follow up.

Table 20

COMPARISON OF BASELINE AND FINAL DIPLOPIA

Gazes	Baseline (%)	Final (%)
Primarygaze	12	10
Upgaze	16	12
Downgaze	6	10
Dextroversion	6	4
Levoversion	4	3
No diplopia	56	57

This table shows that there was reduction in diplopia in the follow up period.

Table 21

COMPARISON OF OCULAR MOTILITY AT BASELINE AND FINAL FOLLOW UP

Restricted motiity	Baseline (%)	Final (%)
Upgaze & downgaze	**20**	**14**
Upgaze	**54**	**22**
Downgaze	**38**	**14**
Abduction	**16**	**4**
Adduction	**8**	**5**
No restriction	**42**	**66**

There was improvement in ocular motility during final followup.

Table 22

CHANGE OF PERIORBITAL ANAESTHESIA

Baseline findings	Improved to normal	Improved	Stayed same	Total
Supraorbital	4	5	0	9
Infraorbital	11	28	4	43
Upper alveolus	0	3	0	3
Others	3	0	0	3
Normal	42	0	0	42
Total	60	36	4	100

This table shows that infraorbital anaesthesia was most common in orbital fractures.

DISCUSSION

Kamath et al did a prospective study on 35 cases of orbital fractures from 1995 to 2005. He analysed the factors like demographics, causes of injury, associated ocular injuries and about surgical management.

Brain S.Beisman[5] et al studied about diplopia after surgical repair of blowout fracture.

Gillbard[30] et al did an analytical study on 19 patients with blowout fracture. He related the CT appearance on coronal sections with clinical presentation.

Herbert S Greenwaldt[36] did a study on orbital fractures with 128 patients. The factors which he analysed included demographics, various causes of injury, indications for surgical treatment and technique selected for repair.

Leib Sohn[51] et al correlated the prolapse of tissue into maxillary antrum with late diplopia and enophthalmos in a study of 365 patients with orbital floor fracture.

Hwang K et al did an analytical study on 391 patients with orbital fractures from 1996 to 2008. He studied about the demographics, common bones involved in orbital fractures, implants used in surgical management and postoperative complications.

Gosau M et al did a prospective study on 189 patients with orbital fractures from 2003 to 2007 about the cause of injury, surgical management and postoperative complications.

Tong & Lawrence did a prospective study on 199 patients over 10 years. The factors he studied included cause of injury, types of fracture and clinical features.

Sang Hun Lee et al did a prospective study on 45 eyes of orbital fractures. He studied about the mean age of onset of orbital fractures and surgical management.

According to Geoffray M K Witko study, most orbital fracturs occur in teenagers or young adults[2] and most cases are managed on outpatient basis.[76,84]

Mark S Brown et al did an analytical study on 400 patients of orbital fractures. According to his study, motor vehicle accidents was the most common cause of injury and also the most common bone involved was floor[82] fracture.

In all these series, the most frequently affected age group is young adults between 21 to 30 years which correlates with our study. Majority of fractures in our study group patients occur between 21 to 30 years.

Males are more frequently involved in all study groups which is also comparable with our findings.

Right eye is more frequently involved than left eye in our study group.

In most of the studies, violence or sport activities account for most of the orbital fractures followed by road traffic accidents. But in Tong et al, Geoffroy M K Witko and Mark S Brown study motor vechicle accidents accounts for majority of the cases which correlates with our study. In this study road traffic accidents accounts for majority of injury followed by violence.

In most of the studies floor fracture were common followed by medial wall fracture except in Sahng's series[79] in which medial wall involvement is most frequent. In this study floor fracture is more common followed by medial wall fracture.

The most common muscle entrapped in orbital fracture is inferior rectus followed by medial rectus.

In our study maxillary sinus involvement was seen in floor fracture. The most common radiological feature is maxillary sinus haziness, followed by fluid level in maxillary sinus and tear drop sign.

Almost equal incidence of pure and impure blowout fracture is seen this study group.

The most common clinical presentation was periorbital ecchymosis followed by extraocular movement restriction.

Associated ocular injuries like hyphema, iridocyclitis, traumatic cataract, globe rupture and choroidal tear were common in this study group.

According to this study, hypertropia was the most common ocular deviation associated with orbital fractures. In most of the eyes, primary position was maintained.

In forced duction test, elevation restriction was present in most of the eyes followed by restriction of abduction.

Enophthalmos was most commonly associated with floor fracture followed by floor and medial wall fracture.

Visual acuity of 6/6 was maintained in most of the cases.(46.2%)

Most of the case were managed medically. The surgical treatment was done mainly for blowout fractures of the orbit. The approach preferred was infraorbital approach.

CONCLUSION

From this prospective study of orbital fractures, the following inferences have been noted.

1) The mean age was 26 years.
2) Male female ratio was 10.2:1.
3) Motor vehicle accidents followed by violence were the most common causes.
4) Orbital floor was frequently involved in orbital fracture followed by medial wall.
5) Incidence of pure blowout fracture and impure blowout fracture were of almost equal frequency.
6) Most common clinical feature encountered was maxillary sinus haziness.
7) Enophthalmos was encountered in 22% of cases and exophthalmos was encountered in 6% of the cases.
8) Most common limited extraocular movement limitation was elevation.
9) Common clinical presentation was periorbital ecchymosis.
10) 46.2% of the patients had normal vision and 5.3% of patients had visual loss.

11) 17 cases underwent surgery. Most common indication of surgery was radiological evidence of muscle entrapment.

12) Most common surgical approach was infraorbital approach.

13) Implants were used in most of the cases of blowout fracture.

14) Good recovery of diplopia and enophthalmos was noted in cases of early surgical intervention.

15) Surgery did not influence the incidence of late enophthalmos or status of infraorbital anaesthesia.

FIG 1. DEMOGRAPHICS

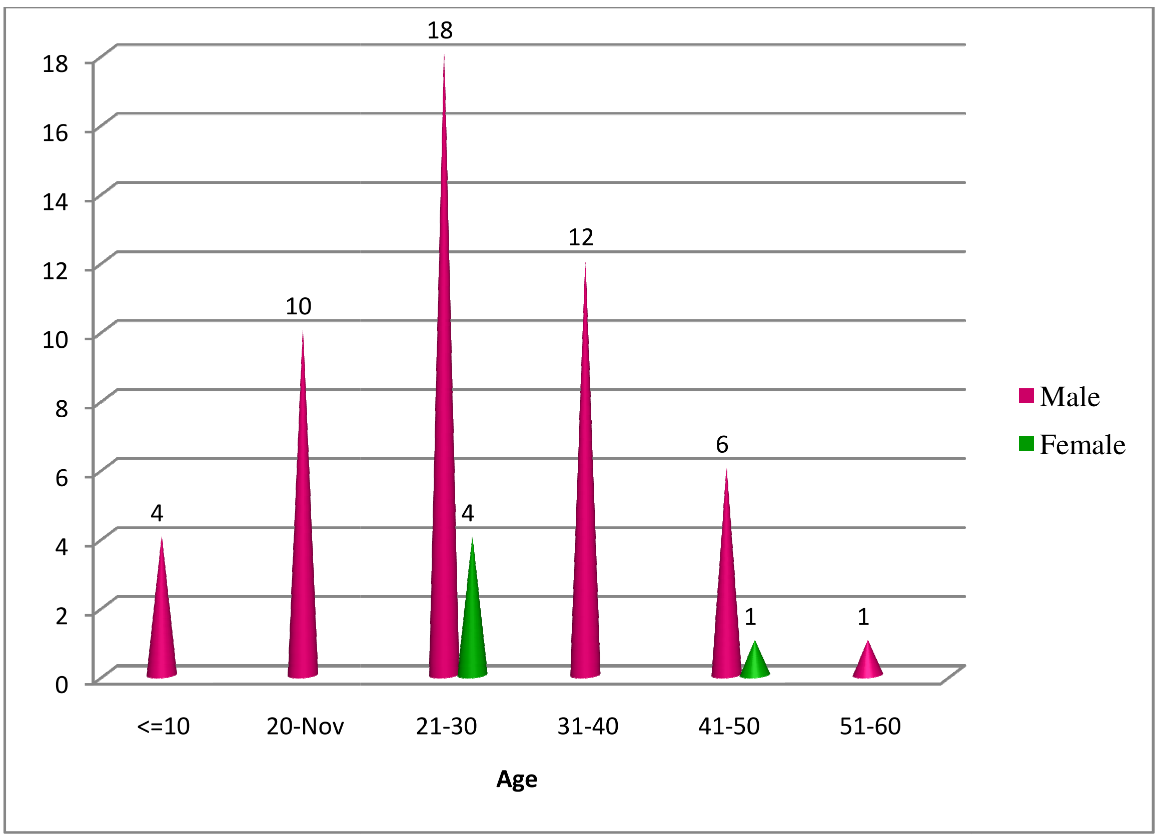

FIG 2. LATERALITY

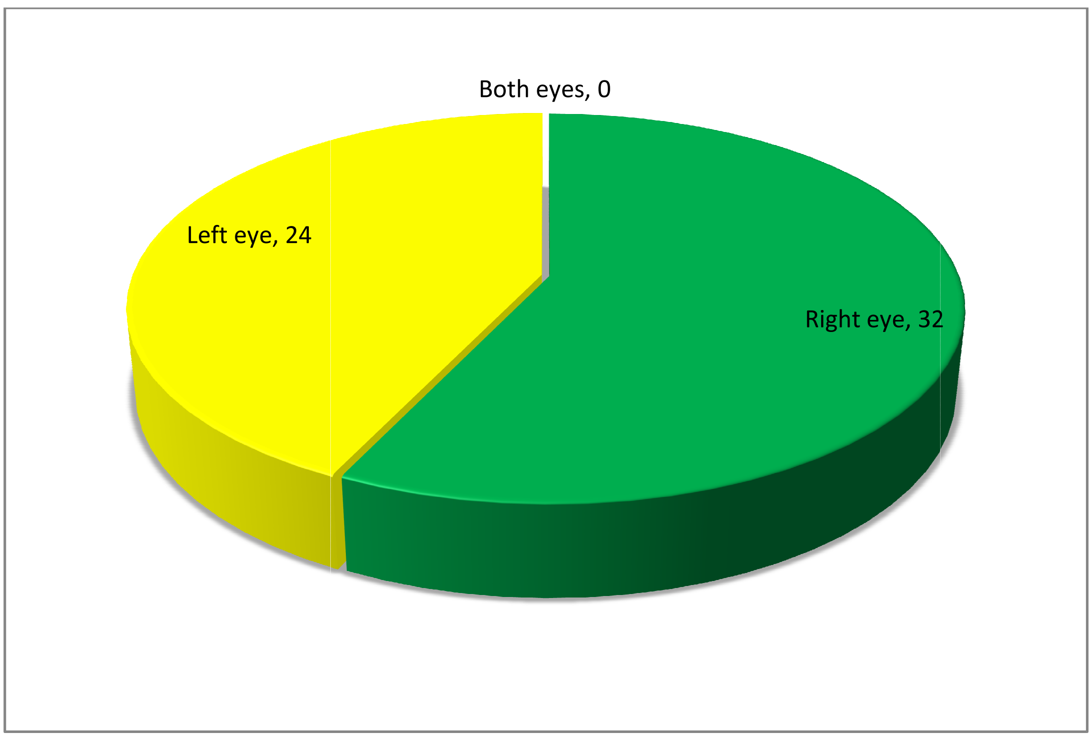

Fig : 3 CAUSES OF INJURY

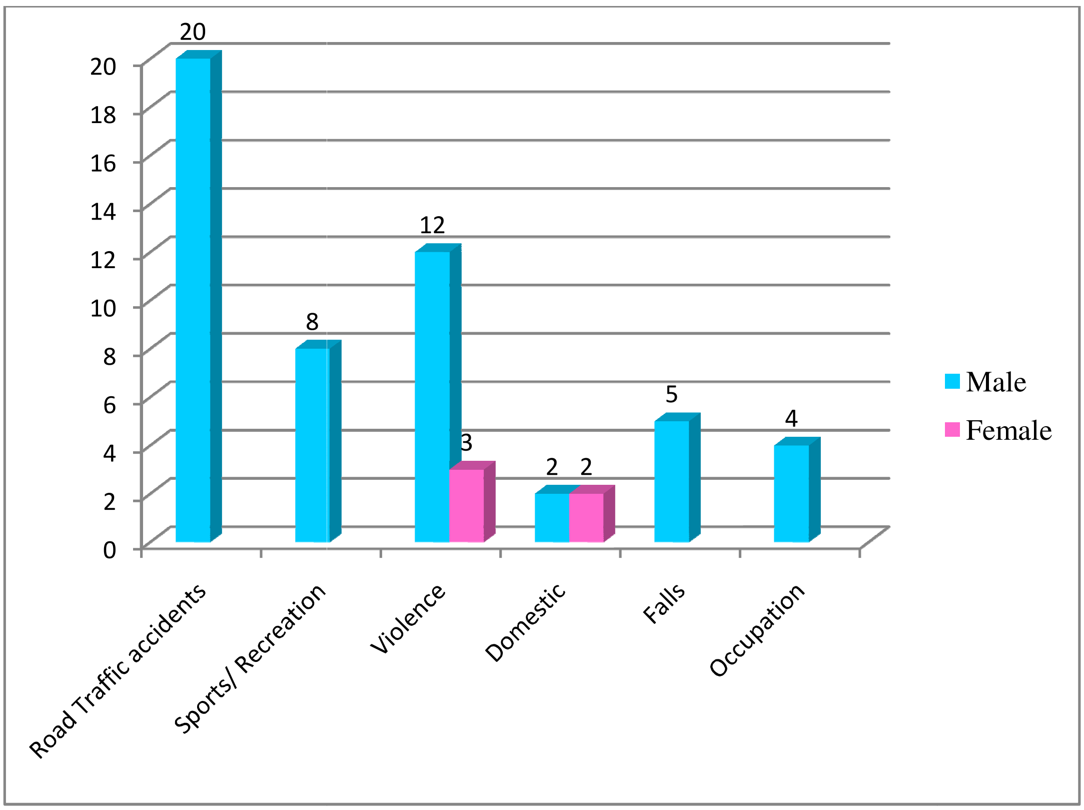

FIG : 4 CLASSIFICATION OF ORBITAL FRACTURES

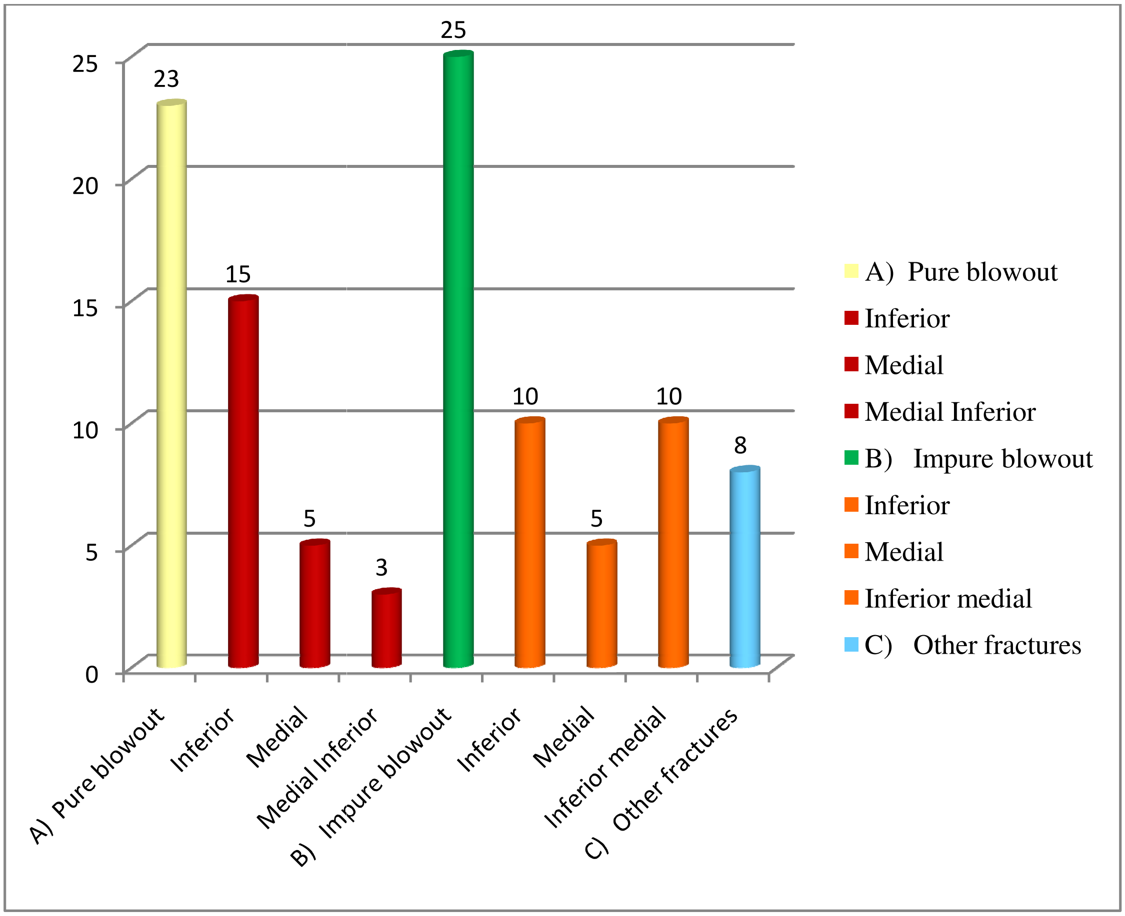

FIG : 5 SITES OF ORBITAL FRACTURES

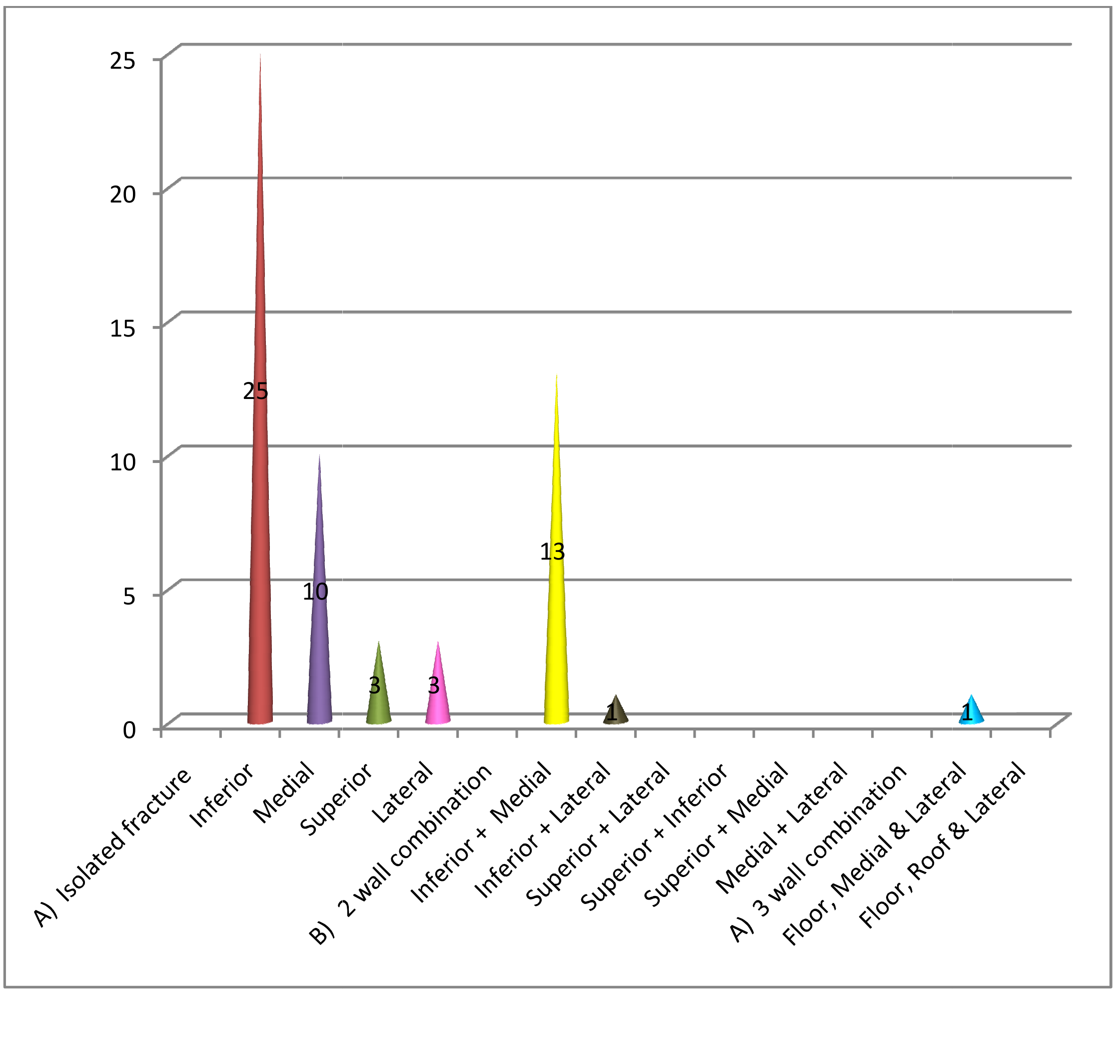

FIG : 6 BASELINE OCULAR DEVIATION

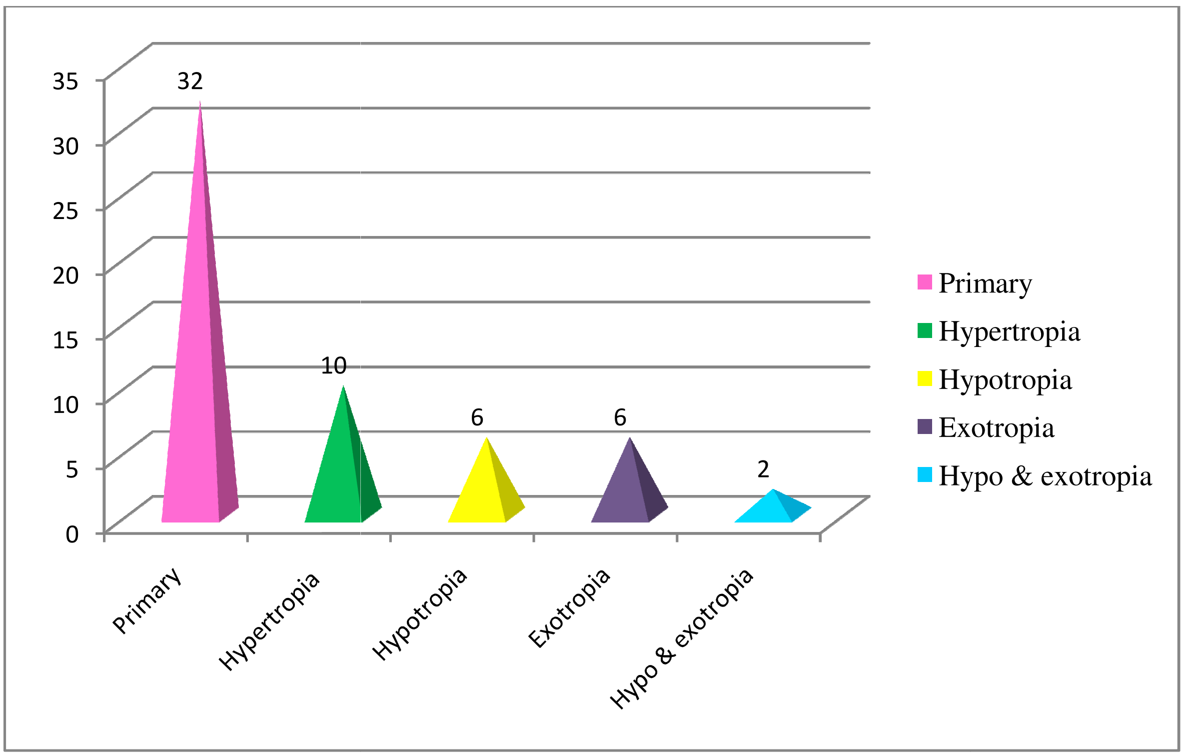

FIG 7. EXTRAOCULAR TISSUE ENTRAPMENT

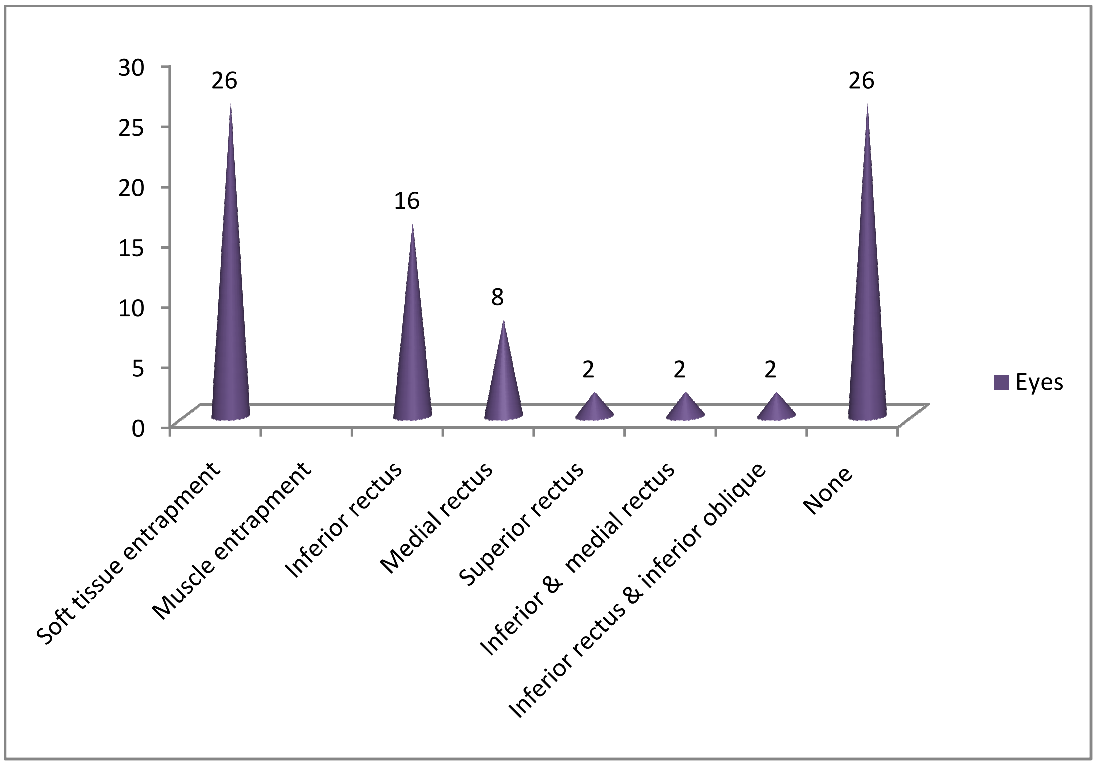

FIG 8 SINUS INVOLVEMENT

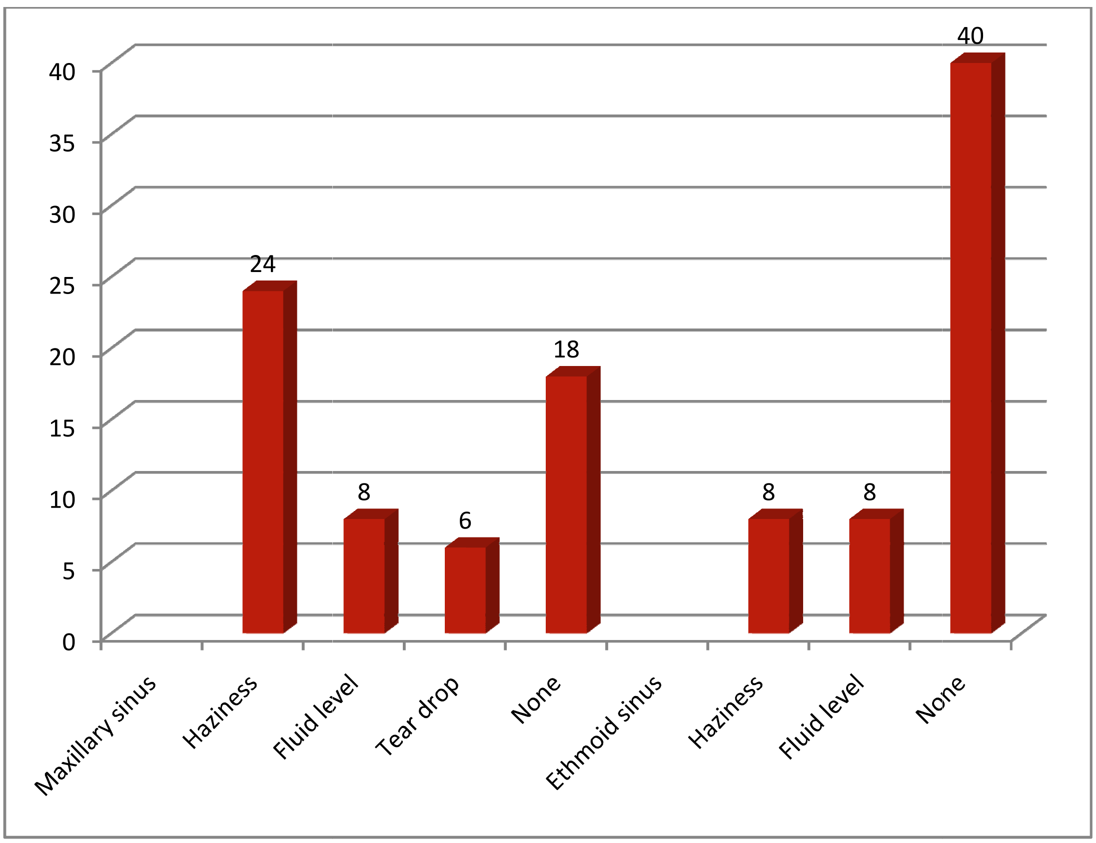

FIG 9 BASELINE FORCED DUCTION TEST

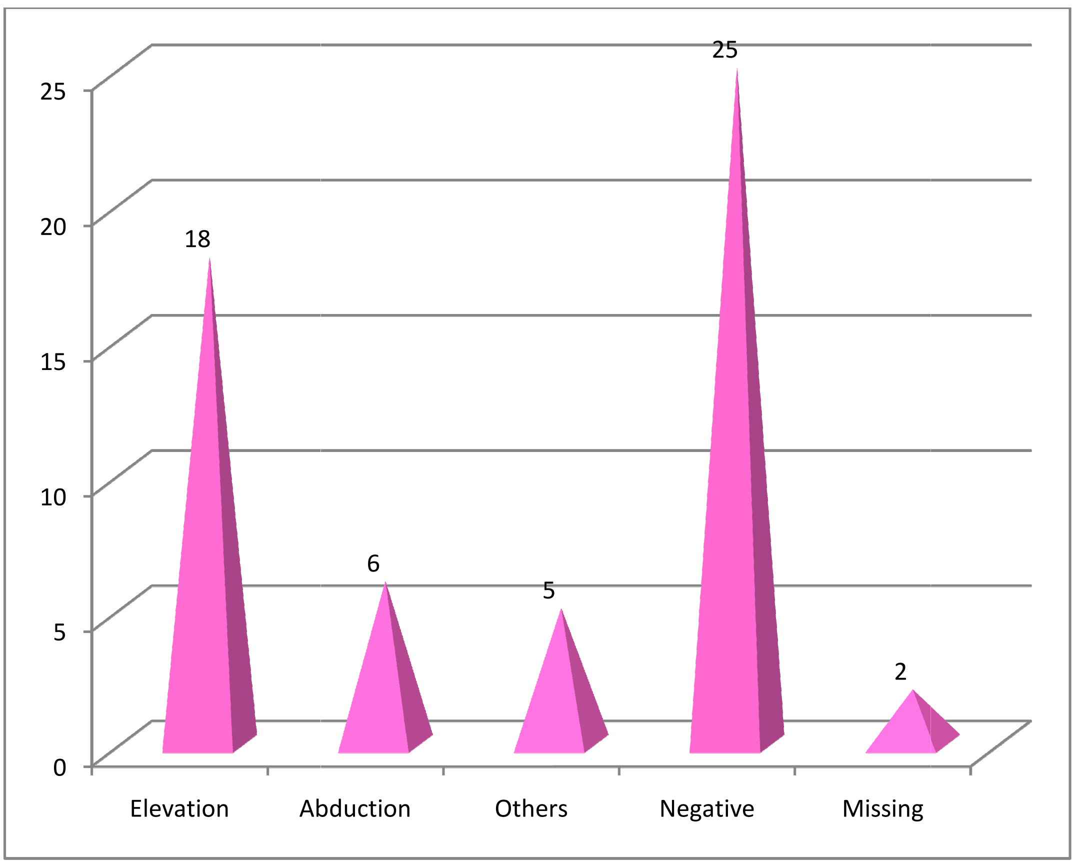

FIG : 1 STRUCTURES PASSING THROUGH SUPERIOR ORBITAL FISSURE

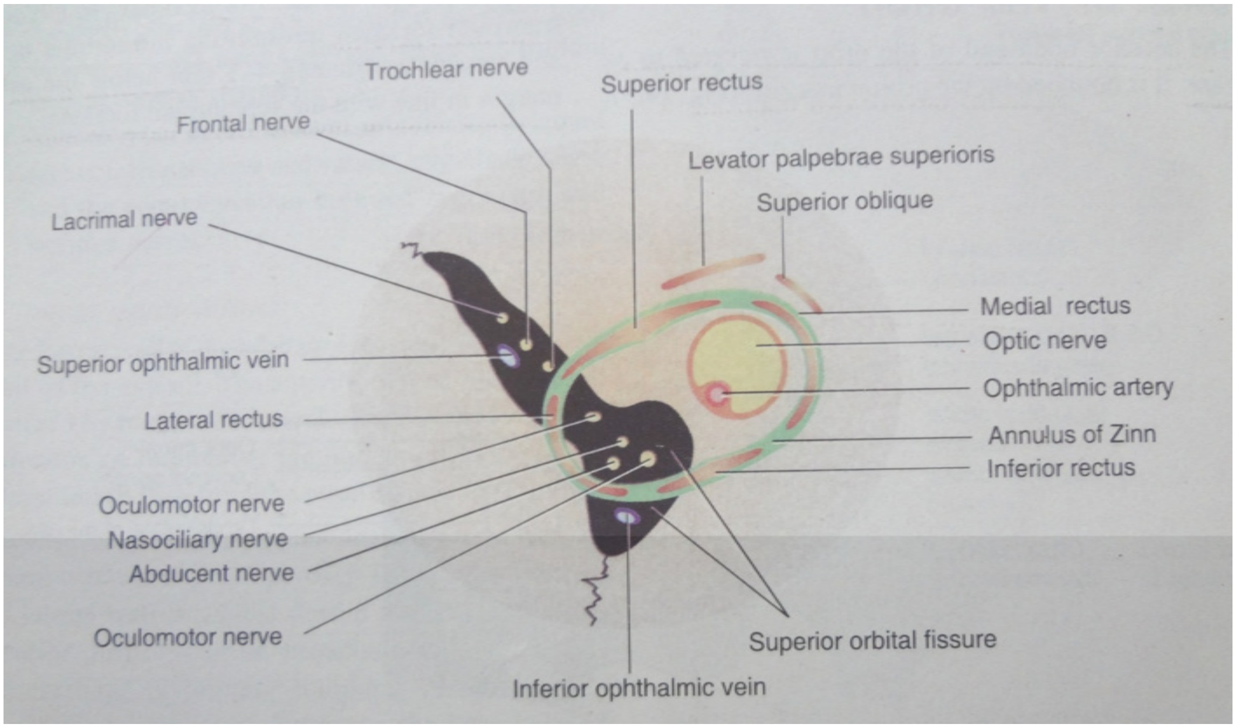

FIG : 2 MEDIAL WALL OF THE ORBIT

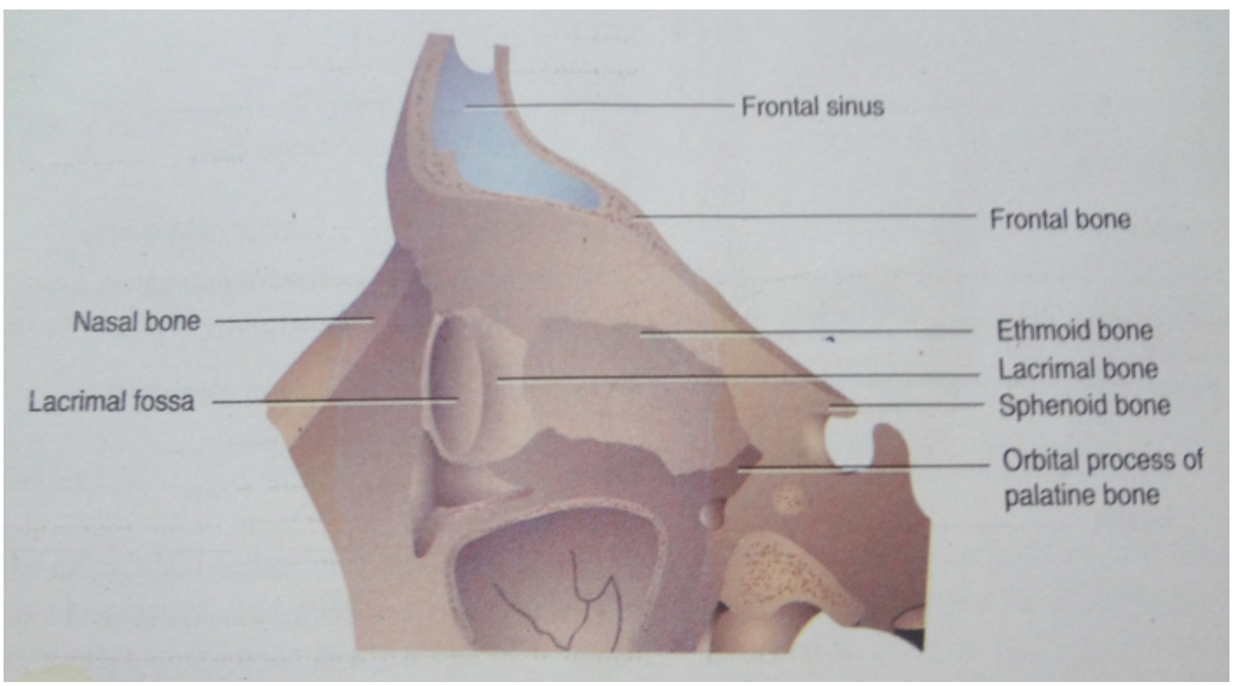

FIG : 3 LATERAL WALL OF THE ORBIT

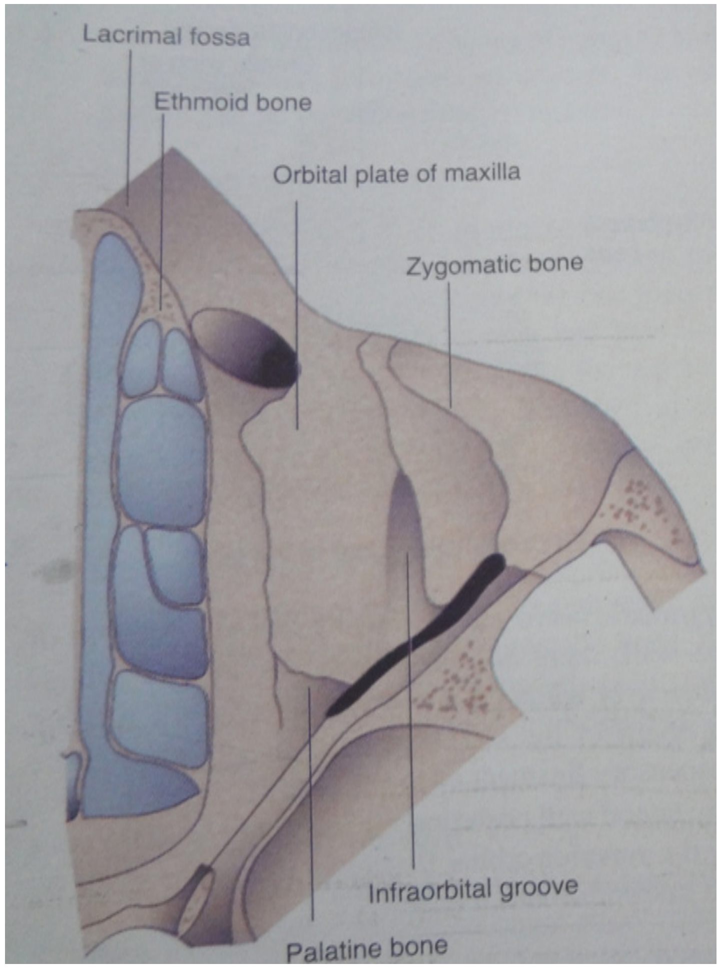

FIG : 4 FRACTURE FLOOR WITH TEAR DROP SIGN

FIG : 5 FRACTURE MEDIAL ORBITAL WALL WITH EMPHYSEMA

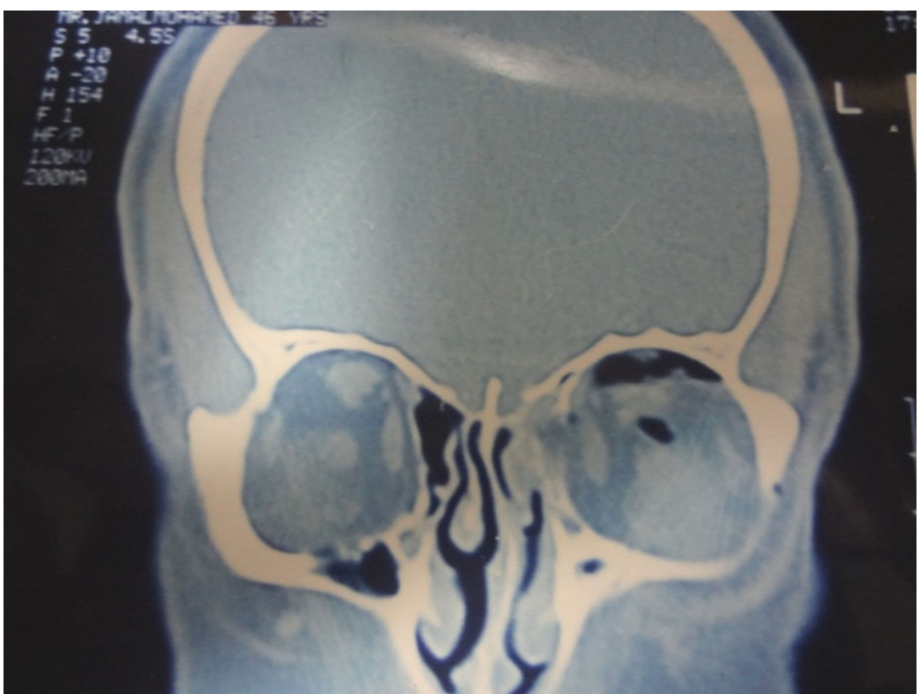

FIG : 6 BLOWOUT FRACTURE ORBIT WITH ENOPHTHALMOS – LEFT EYE

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DATA COLLECTION PROFORMA

Name

Age

Address

OP & IP NO

Date of admission

Sex

Occupation

Income

Complaints

Date of injury

Cause of injury: [1] RTA [2] Assault [3] Fall [4] Sports [5]Recreation [6]Domestic [7]School

Mechanism of injury

Objects involved

H/O defective vision RE/LE

Duration

Onset

Severity

Course

H/O diplopia

Time of onset

Separation of images

Unilateral/ bilateral

H/O pain

Site

Duration

Radiating to

Aggravating and relieving factors

H/O Epistaxis

H/O Redness : RE/LE

Watering

Itching

Photophobia

Pain

H/O loss of consciousness:

Any other relevant history

Past history :

Diabetes/hypertension/tuberculosis/asthma/alcoholic/smoker

General examination:

Vitals

Pulse

Blood pressure

Respiratory system

Cardiovascular system

Local examination

Inspection

Forehead

Eyebrow

Lid RE/LE – normal/contused/tear/echymosis/edema/crepitus/emphysema/lagophthalmos/ ptosis/scar

Periorbita

Enophthalmos

Ocular motility : normal/ restricted in adduction, abduction, elevation, depression

Hertel'sexophthalmometer

Palpation

Orbital margin- irregularity,gaps,step deformity, tenderness,

Anterior segment examination: RE LE

Lids

Conjunctiva

Cornea

Anterior chamber

Iris

Pupil : size, shape, reaction to light-direct/consensual/accomodation

lens

Hess Chart

Diplopia chart

Forced duction test

Investigation

X-ray orbit

CT-orbit

MRI Orbit

Ref. No. 3104/E4/3/2012

Govt.Rajaji Hospital,Madurai.20.
Dated: .03.2012

Institutional Review Board / Independent Ethics Committee.
Dr. A. Edwin Joe, M.D (FM), BL.,
Dean, Madurai Medical College & 2521021 (Secy)
Govt Rajaji Hospital, Madurai 625020.
Convenor
grhethicssecy @gmail.com.

Sub: Establishment-Govt. Rajaji Hospital, aMadurai-20-
Ethics committee-Meeting Agenda-communicated-regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 11.00 Am to 1.00Pm on 29.03.2012 at the Dean Chamber, Govt. Rajaji Hospital, Madurai. The following members of the committee have been attended the meeting.

----

1. Dr.N.Vijayasankaran,M.ch(Uro.) 094-430-58793 0452-2584397	Sr.Consultant Urologist Madurai Kidney Centre, Sivagangai Road,Madurai	Chairman
2. Dr.P.K. Muthu Kumarasamy, M.D., 9843050911	Professor & H.O.D of Medical, Oncology(Retired)	Member Secretary
3. Dr.T.Meena,MD 094-437-74875	Professor of Physiology, Madurai Medical College	Member
4. Dr. S. Thamilarasi, M.D (Pharmacol)	Professor of pharmacology	
5.Dr.Moses K.Daniel MD(Gen.Medicine) 098-421-56066	Professor of Medicine Madurai Medical College	Member
6.Dr.M.Gobinath,MS(Gen.Surgery)	Professor of Surgery Madurai Medical College	Member
7.Dr.S. Dilshadh, MD(O&G) 9894053516	Professor of OP&Gyn Madurai Medical College	Member
8.Dr.S.Vadivel Murugan., M.D, 097-871-50040	Professor of Medicine Madurai Medical College	Member
9.Shri.M.Sridher,B.sc.B.L. 099-949-07400	Advocate, 2, Deputy collectors colony 4th street KK Nagar, Madurai-20.	Member
10.Shri.O.B.D.Bharat,B.sc., 094-437-14162	Businessman Plot No.588, K.K.Nagar,Madurai.20.	Member
11.Shri. S.sivakumar,M.A(Social) Mphil 093-444-84990	Sociologist, Plot No.51 F.F, K.K Nagar, Madurai.	Member

Following Projects were approved by the committee

Sl. No	Name of P.G.	Course	Name of the Project	Remarks
1.	Nithya. R	PG, M.S (Ophthal)	Clinical study of the Management of orbital fractures	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.
4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

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All the above members and Head of the Departments concerned.
All the Applicants.

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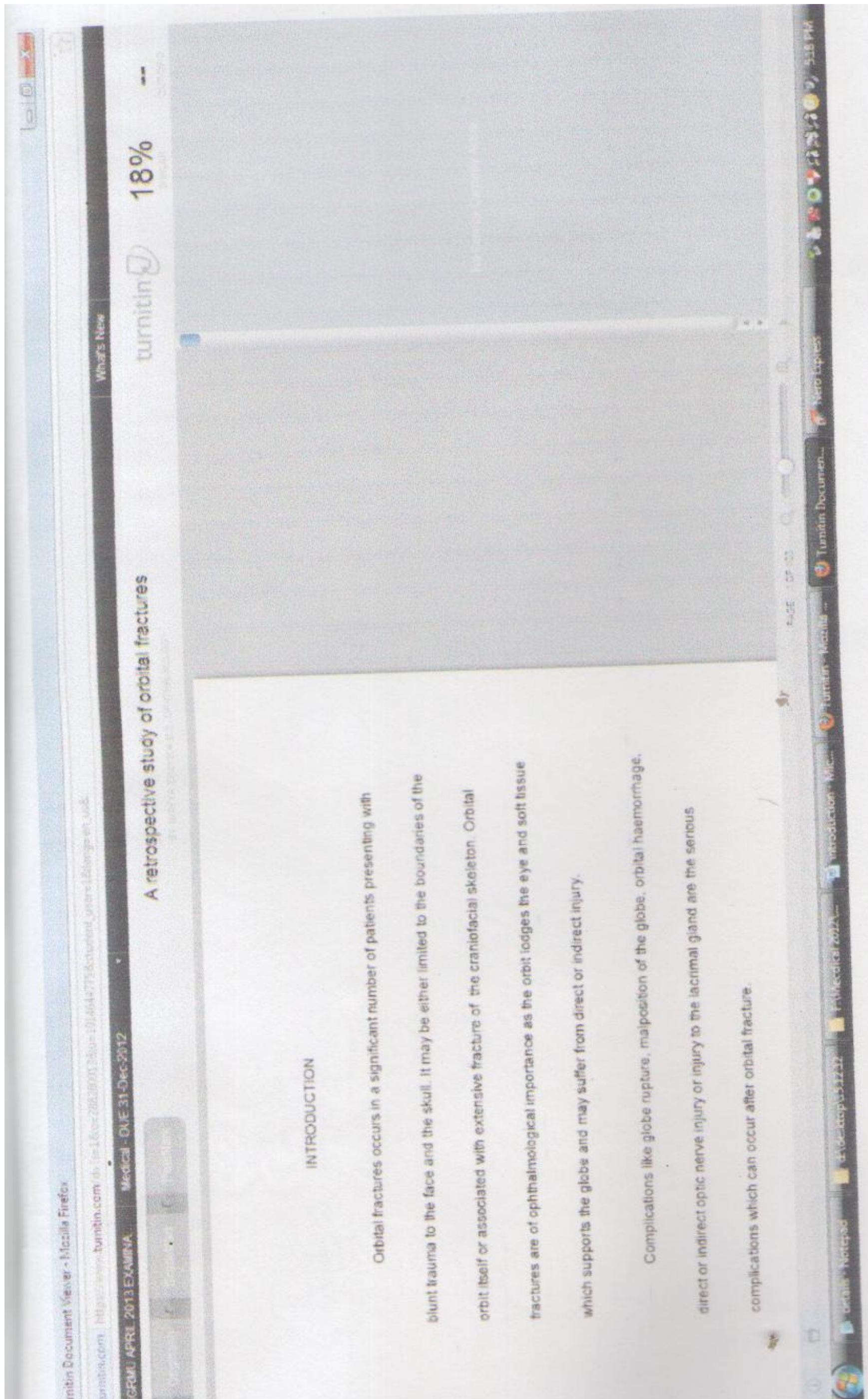
INTRODUCTION Orbital fractures occurs in a significant number of patients presenting with blunt trauma to the face and the skull. It may be either limited to the boundaries of the orbit itself or associated with extensive fracture of the craniofacial skeleton. Orbital fractures are of ophthalmological importance as the orbit lodges the eye and soft tissue which supports the globe and may suffer from direct or indirect injury. Complications like globe rupture, malposition of the globe, orbital haemorrhage, direct or indirect optic nerve injury or injury to the lacrimal gland are the serious complications which can occur after orbital fracture. The long term sequelae of orbital fractures are...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INTRODUCTION
Orbital fractures occurs in a significant number of patients presenting with blunt trauma to the face and the skull. It may be either limited to the boundaries of the orbit itself or associated with extensive fracture of the craniofacial skeleton. Orbital fractures are of ophthalmological importance as the orbit lodges the eye and soft tissue which supports the globe and may suffer from direct or indirect injury.
Complications like globe rupture, malpositon of the globe, orbital haemorrhage, direct or indirect optic nerve injury or injury to the lacrimal gland are the serious complications which can occur after orbital fracture.

MASTER CHART

S.No	Name	Age	I.P.No	Sex	Laterality	Causes	Bone Involved	Visual Acuity	Diplopia	Enophthalmos	Restricted motility	Infraorbital anaesthesia	Anterior Segment	Management
1	Murugan	22	54234	M	RE	RTA	Floor	6/12	No	No	Upgaze	Yes	Normal	Medical
2	Kannan	23	54678	M	LE	Sports	Floor	6/12	Upgaze	Yes	Upgaze	No	Normal	Surgical
3	Raja	19	56745	M	RE	Sports	Medial	6/12	Levoversion	No	Abduction	No	Normal	Medical
4	Muthu	29	57867	M	RE	RTA	Roof	6/9	Upgaze	No	Downgaze	No	Iridocyclitis	Medical
5	Kumar	12	56782	M	LE	Falls	Floor	6/60	No	No	Upgaze	Yes	Normal	Medical
6	Prabhu	33	56790	M	RE	Occupation	Lateral	6/12	Dextroversion	Yes	Adduction	No	Normal	Medical
7	Krishnan	24	57890	M	RE	Violence	Floor	6/12	Primary gaze	No	Upgaze	Yes	Normal	Medical
8	Manickam	35	58765	M	LE	Falls	Inf+Medial	PL	No	No	Up&Downgaze	No	Normal	Surgical
9	Malaisamy	17	57643	M	LE	RTA	Floor	5/60	Upgaze	No	Upgaze	Yes	Hyphema	Surgical
10	Meenakshi	26	50987	F	RE	Violence	Inf+Medial	6/12	No	No	Up&Downgaze	Yes	Normal	Medical
11	Palanisamy	14	67894	M	LE	Sports	Medial	6/12	No	No	Abduction	No	Normal	Medical
12	Kandasamy	44	68903	M	RE	RTA	Medial	3/60	Dextroversion	Yes	Abduction	Yes	Iridocyclitis	Medical
13	Manikandan	35	67345	M	LE	Falls	Floor	6/12	Upgaze	No	Upgaze	Yes	Normal	Medical
14	Malar	28	67021	F	RE	Violence	Roof	6/12	No	Yes	Downgaze	Yes	Normal	Medical
15	Muthusamy	46	69043	M	LE	RTA	Floor	3/60	Primary gaze	Yes	Upgaze	No	Lens subluxation	Medical
16	Thangavel	22	57891	M	RE	RTA	Medial	6/12	No	No	Abduction	No	Normal	Medical
17	Pachiappan	45	50934	M	RE	Violence	Inf+Medial	1/60	No	No	Up&Downgaze	Yes	Lens dislocation	Surgical
18	Sethu	5	50666	M	LE	RTA	Medial	6/12	Primary gaze	Yes	Abduction	No	Normal	Medical
19	Seetha	25	50992	F	RE	Domestic	Inf+Medial	No PL	Dextroversion	No	Upgaze	No	RAPD	Medical
20	Sivaraman	36	59330	M	LE	RTA	Inf+Medial	6/12	Dextroversion	No	Upgaze	No	Normal	Medical
21	Kanchana	45	55589	F	RE	Violence	Inf+Medial	No PL	No	Yes	Up&Downgaze	Yes	Hyphema	Surgical
22	Jeganathan	45	55521	M	LE	RTA	Floor	6/60	Primary gaze	Yes	Upgaze	No	Traumatic cataract	Surgical
23	Mahendran	46	66789	M	RE	Occupation	Roof	6/9	Down gaze	No	Downgaze	No	Normal	Medical
24	Devarajan	16	65543	M	RE	Falls	Inf+Medial	6/9	Upgaze	No	Upgaze	Yes	Normal	Medical

25	Sharathi	48	68890	M	RE	RTA	Floor	5/60	No	No	Upgaze	No	Lens subluxation	Surgical
26	Mathavan	27	67789	M	RE	Sports	Floor	6/9	No	Yes	Upgaze	Yes	Normal	Medical
27	Karupaiya	18	61123	M	LE	Occupation	Floor	6/9	Primary gaze	No	Upgaze	No	Normal	Medical
28	Anand	25	65578	M	RE	Violence	Medial	5/60	Dextroversion	No	Abduction	Yes	Traumatic cataract	Surgical
29	Arul Guhan	26	60098	M	LE	RTA	Floor	3/60	No	No	Upgaze	No	Traumatic cataract	Surgical
30	Saminathan	26	63325	M	LE	Sports	Floor	PL	No	No	Upgaze	Yes	Hyphema	Medical
31	Manavalan	35	68890	M	RE	RTA	Floor	6/60	Upgaze	Yes	Upgaze	No	Iridocyclitis	Medical
32	Natarajan	55	65554	M	RE	Violence	Medial	6/6	Primary gaze	No	Abduction	Yes	Normal	Medical
33	Nachiappan	24	69990	M	RE	RTA	Floor	1/60	Primary gaze	No	Upgaze	No	RAPD	Medical
34	Thangadurai	37	63345	M	LE	Violence	Floor	6/6	No	No	Upgaze	Yes	Normal	Medical
35	Thendral	8	55555	M	RE	RTA	Floor	6/6	Upgaze	Yes	Upgaze	Yes	Normal	Medical
36	Anbumani	27	55566	M	RE	Sports	Lateral	6/6	No	No	Adduction	No	Normal	Surgical
37	Chitra	27	55577	F	LE	Domestic	Inf+Medial	PL	Primary gaze	Yes	Upgaze	No	RAPD	Medical
38	Chinnadurai	18	58999	M	RE	RTA	Floor	6/6	No	Yes	Upgaze	No	Iridocyclitis	Medical
39	Pandiyan	16	66678	M	RE	Violence	Inf+Medial	6/6	Dextroversion	Yes	Upgaze	Yes	Iridodialysis	Surgical
40	Andavar	36	63345	M	LE	RTA	Medial	6/6	No	No	Abduction	No	Normal	Medical
41	Marimuthu	26	66645	M	RE	Violence	Inf+Medial	6/6	Primary gaze	Yes	Abduction	No	Iridodialysis	Surgical
42	Sengodan	34	66334	M	RE	RTA	Floor	6/12	Upgaze	No	Upgaze	No	Normal	Medical
43	Govindan	27	66098	M	LE	Violence	Inf+Medial	6/12	No	Yes	Upgaze	Yes	Normal	Medical
44	Vadivel	34	44567	M	RE	Violence	Medial	6/6	No	No	Abduction	Yes	Normal	Surgical
45	Arasu	19	45689	M	LE	RTA	Floor	6/6	Upgaze	No	Upgaze	No	Iridocyclitis	Surgical
46	Venkatesan	25	44367	M	RE	Sports	Inf+Medial	6/6	No	Yes	Upgaze	No	Normal	Surgical
47	Velayutham	37	43567	M	RE	Violence	Medial	6/9	No	No	Abduction	No	Normal	Medical
48	Arulmani	28	44390	M	RE	RTA	Floor	3/60	Primary gaze	No	Upgaze	Yes	Traumatic cataract	Surgical
49	Vijay	8	43278	M	LE	Domestic	Inf+Lateral	5/60	Levoversion	Yes	Upgaze	No	Iridodialysis	Medical
50	Gangatharan	38	44890	M	RE	RTA	Floor	6/6	No	Yes	Upgaze	Yes	Normal	Medical
51	Ganapathy	18	54400	M	RE	Falls	Floor	6/6	Primary gaze	Yes	Upgaze	No	Normal	Medical
52	Murali	27	49901	M	LE	Violence	Floor	6/6	No	Yes	Upgaze	Yes	Traumatic cataract	Surgical
53	Arjun	9	44690	M	LE	Domestic	Lateral	6/9	Levoversion	No	Upgaze	No	Normal	Medical
54	Senthil	24	44553	M	LE	Sports	Floor	6/9	Upgaze	Yes	Upgaze	Yes	Normal	Surgical
55	Agasthian	36	44434	M	RE	Occupation	Inf+Medial	6/9	No	No	Upgaze	Yes	Normal	Medical
56	Akilan	27	44888	M	RE	Violence	Floor+Med+Lat	No PL	No	No	Up&Downgaze	No	Iridodialysis	Medical